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NEPHROPATHIES

THE classification of disease is of much more than academic interest, and serves different purposes The classification will, therefore, be approached from different viewpoints, and it is often impossible to harmonize the results This is especially true in the clinical, etiologic, pathologic, and functional classifications of the diseases of the kidney Bright's disease, in its original significance, is of little more than historic interest, as much more is now included in the name than "a disease of the kidney associated with dropsy and albuminuria."

Until the 90's the structural criteria were the main bases for the classification, and here, as Aschoff says, every pathologist has his own "Glaubens bekenntnis" The correlation of clinical and pathologic findings, using the old terms "parenchymatous" and "interstitial nephritis," has notoriously failed, as has been realized by all clinicians who have followed their cases to autopsy

In later times much more interest has attached to the study of function, and efforts have been made to correlate the functional with the anatomic findings—Schlayer, Widal, and others These attempts cannot be said to have been successful so far as concerns the human nephropathies There has, however, arisen out of these studies a closer association of the clinician, pathologic anatomist, and pathologic physiologist, which has markedly advanced our knowledge of the nephropathies, and evolved a

classification which works better from the standpoint of prognosis, and harmonizes more nearly the clinical and pathologic findings

The best classification seems to me to be that of Volhard and Fahr, in their monograph, "Die Brightische Nierenkrankheit" Similar classifications have been proposed by Dr Barker, in Monographic Medicine, Vol III, 1916, and by Dr Stengel, in MEDICAL CLINICS OF NORTH AMERICA, September, 1917 Volhard and Fahr's classification is as follows

A. Degenerative diseases Nephrosis, genuine and known etiology, with and without amyloid degeneration of the vessels.
- I Acute course
- II Chronic course
- III End stage Nephrotic contracted kidney without increased blood-pressure.

Subdivision Necrotic nephroses

B Inflammatory diseases Nephritides
1. Diffuse glomerulonephritis, with obligatory increased blood pressure. Course in three stages
   - I. The acute stage
   - II The chronic stage, without kidney insufficiency
   - III The end stage, with kidney insufficiency

   All three stages may run their course (*a*) without marked edema, (*b*) with marked edema, *i. e.*, with marked and diffuse degeneration of tubular epithelium (mixed form)
2. Focal nephritis, without raised blood pressure
   - (*a*) Focal glomerulonephritis
     - I. Acute stage.
     - II. Chronic stage
   - (*b*) Septic interstitial focal nephritis
   - (*c*) Embolic focal nephritis.

C Arteriosclerotic diseases Scleroses
- I The bland, favorable "hypertonie" = pure sclerosis of the kidney vessels
- II The combination form Malignant genuine contracted kidney = sclerosis plus nephritis

I desire to present a series of cases which we may classify clinically according to this scheme

CASE I —S M , seventeen years of age, male, negro, laborer in a levee camp *Nephrosis, lues*

Anamnesis —Family history negative Had measles at ten years of age Pertussis in childhood Never had scarlet fever or diphtheria He had "chills and fever" in 1912 He has frequently had sore throat, but does not remember having a severe

attack Has never had rheumatism Had an ulcer on penis last year, does not think that it was followed by sore mouth or eruption over the body Took no treatment for same.

He was admitted to Ward E, Memphis General Hospital, October 31, 1917, complaining of general swelling of his body and shortness of breath About two weeks before admittance he was much exposed to cold and wet, incident to his work as a laborer in a levee camp At this time he noticed swelling of his legs and feet, shortness of breath, diarrhea, and general weakness The swelling gradually increased until it involved the whole body

Status Præsens.—November 1, 1917 A well-developed negro boy, about seventeen years of age. Mucous membranes of good color There is a marked generalized edema involving the face, neck, trunk, extremities, and external genitals, signs of fluid in the right pleural cavity, râles over both lungs, front and back

Heart—P M I. in the fifth interspace, 8 cm. from the mid-sternal line, area of cardiac dulness not enlarged, at apex faint systolic murmur, not transmitted, pulmonic second sound not accentuated

Abdomen—Ascites, liver nor spleen palpable. Nervous system negative

Urine—Daily quantity not measured, but scant. November 6, 1917 Color, dark brown, sp gr , 1020, reaction, alkaline, a large quantity of albumin, no sugar, casts present, no red cells or pus cells

Blood-pressure—Systolic, 115, diastolic, 70 (auscultatory)

Blood (11/6/17)—Leukocytes, 8000, polymorphonuclears, 58 per cent , small lymphocytes, 36 per cent., large mononuclears, 5 per cent., eosinophils, 1 per cent. 11/12/17 Erythrocytes, 5,182,000, leukocytes, 9000, polymorphonuclears, 66 per cent., small lymphocytes, 31 per cent., large mononuclears, 0, eosinophils, 3 per cent. Wassermann reaction positive (++++)

Functional Tests

MOSENTHAL KIDNEY TEST-DIET, 11/8/17

Time of day	Urine. C.c.	Sp gr	NaCl Per cent.	NaCl Gm.	N Per cent.	N Gm.
8–10	70	1020				
10–12	60	1020				
12– 2	62	1018				
2– 4	72	1020				
4– 6	87	1020				
6– 8	74	1020				
Total day	425		1	.425	1 5	6 37
Night, 8–8	600	1016	04	24	1 0	6 00
Total, 24 hours	1025			665		12 37
Intake fl , NaCl, and N	1760			8 5		13-4
Difference	+735			+7 25		+1 03

Weight.	Date	Period	Blood. Mgm urea per 100 c.c.	Period	24 hrs	Urine. Urea	U p l.	Ambard
57	11/8/17	4–6	40	87 c.c	1044	18 5	17 7	coeff .093

R p H (Marriott), 8 2 Phenolsulphonephthalein (intravenously), 2 hrs , 55

Course —The patient ran an afebrile course, with pulse from 68 to 86 No discomfort except that attributable to the anasarca and the accumulation of fluid in the serous cavities There was no evidence of uremia The anasarca, hydrothorax, and ascites gradually disappeared After a stay of six weeks in the hospital he left 12/14/17 of his own accord The urine on his discharge showed sp gr 1018, a trace of albumin, hyaline and granular casts Blood-pressure, S P 110, D P 65

Degenerative changes, of the nature of cloudy swellings, occur in the epithelium of the kidney tubules as an accompaniment of most infectious diseases, but there are no clinical manifestations of this other than albuminuria and possibly cylindruria More severe degenerative changes occur as a result of certain infectious diseases, such as diphtheria, tuberculosis, syphilis, chronic suppurations, viridans sepsis, measles, possibly typhoid and paratyphoid, as the result of endogenous intoxications, as in pregnanc and in malignant tumors, as the result of mineral poisons, as wit bichlorid of mercury (nephrosis of known cause) Degenerativ changes also occur without discoverable cause (genuine nephrosis)

Clinically these cases show marked general anasarca and transudation into the serous cavities The fluid is of a pseudochylous nature due to globulin lipoid bodies, is watery and poor in albumin The urine is of a grayish yellow or grayish brown color, cloudy, rich in urates, of high specific gravity, diminished in quantity, exceedingly rich in albumin, and microscopically shows casts, but rarely red blood cells, and with the polarizing microscope a double refracting substance (Munk)

The functional capacity of the kidney is little impaired, as shown by the various functional kidney tests There is very little tendency to uremia The blood is not hydremic, but concentrated, showing an increased number of erythrocytes and increased viscosity The blood-serum, like the transudates in the serous cavities, is of a pseudochylous appearance. The skin, in contradistinction to the objective blood findings, is often pale, there is frequently anorexia, and a marked tendency to diarrhea The eye-grounds seldom show any changes, and the general symptoms of discomfort are lacking, except those due to dropsy

The characteristic features of these cases is the absence of increased blood pressure and cardiac hypertrophy In the case presented I desire to call your attention to the presence of lues and the history of exposure to cold and wet as etiologic factors, clinically, to the marked general anasarca, hydrothorax, and ascites—unfortunately the fluid was not examined—to the absence of cardiac hypertrophy, to the large quantity of albumin in the urine and the absence of hematuria, to the blood pressure, 115–70, to the high blood count, 5,182,000, to the absence of uremia, and to the results of the functional kidney tests You will note that there is a high, fixed specific gravity, a slight night polyuria with a fair sp gr , a very low salt excretion and a good nitrogen excretion These results, in the main, agree with the water test, concentration test, and salt and nitrogen excretion in the cases of Volhard and Fahr, which they attribute to presence of the dropsy, due to the increased permeability of the vessels rather than to impaired functional capacity of the kidney The blood urea is 40 mgm per 100 c.c. The Ambard 0 093 slightly increased, and the phthalein 55 per cent. in two hours

The prognosis in these cases is not grave, so far as the results of the kidney condition are concerned Those due to diphtheria and lues frequently run a short course and terminate in recovery Those due to other causes may run a very prolonged course, even if the edema disappears, the characteristic urine persists and the blood-pressure does not rise, rarely the process in the kidney may pass into the end stage, with contraction and impaired functional capacity, the blood-pressure still not being increased

If the patient should come to autopsy, according to Volhard and Fahr, in the edema stages, the kidneys will show the following—macroscopically enlargement, the capsule strips easily, the outer surface is smooth, the substance is more spongy than normal, the cortex is widened and of a smutty appearance, and the medulla is of a brownish color, microscopically the epithelium, especially of the lower end of the proximal convoluted tubules, shows granular and fatty degeration, that of Henle's loops and the distal convoluted tubules may show similar changes, but not marked, and Henle's loop and the collecting tubules may contain casts In the malpighian corpuscles there may be fatty changes in the glomerular epithelium In the capsular space there may be found coagulated albumin and occasionally degenerated epithelium, the glomerular loops are well filled with blood The interstitial tissue may show slight lymphocytic infiltration and the vessels show no changes unless the nephrosis has developed in a patient with arteriosclerosis

In the cases which have passed into the stage where the edema has subsided there will be more evidence of inflammatory reaction in the interstitial tissue, and in the end-stage a small contracted kidney, due to marked overgrowth of fibrous connective tissue without evidence of inflammatory reaction in the glomeruli may be found, which may be associated with impaired kidney function, but not associated with raised blood-pressure and cardiac hypertrophy

CASE II —Mrs ———, thirty years of age, housewife, *bichlorid of mercury poisoning—necrotic nephrosis*

Anamnesis —Family history negative No serious diseases in past history Two children Domestic infelicity

At about 8 P M , May 31, 1916, swallowed 52½ grains bichlorid of mercury—seven 7½ grain tablets dissolved in a glass of water—supposed to have been headache medicine. The patient vomited and complained of pain and burning under the sternum. Her physician was called immediately He reached the patient in about fifteen minutes The stomach was washed out thoroughly with milk. Vomiting continued at intervals through the night and mucosanguinolent discharges from the bowels She voided urine during the night, the last time just before day Patient was admitted to a private ward of St Joseph's Hospital, Memphis, about 5 P M , June 1, 1916

Status Præsens.—A well-developed, highly cultivated lady, in a good state of nutrition, very weak, but anxious to recover The mucous membranes of the mouth and throat very red. She complains of soreness of the throat, difficulty in swallowing, burning under the sternum, and griping pains in the abdomen There were frequent discharges from the bowels—mucus and blood with tenesmus The temperature was 100⅘° F and pulse 110

For nine days there was very great weakness, depression, much nausea and vomiting, frequent bowel movements—six to twenty-seven daily—backache, headache, twitching of the muscles, delirium, hiccup On June 8th there were very severe hemorrhages from the stomach and bowels There was severe salivation and stomatitis There was during this time no edema, later slight puffiness of the face developed

Urine —There was complete anuria from June 1st to 9th, on this day about 150 c.c. was passed It was of a pale, greenish tinge, sp gr 1012, faintly acid reaction, a trace of albumin, the centrifuged specimen contained a few red cells, leukocytes, much degenerated epithelium, broad and narrow, finely granular and epithelial casts. The urine rapidly increased in quantity—6/10, 450 c.c., 6/11, 1190 c.c., 6/12, 1395 c.c., 6/13, 1500 c.c., 6/14, 2580 c.c., 6/15, 2250 c.c., 6/16, 1650 c c , and continued at about that quantity until discharge On 6/16 phenolsulphonephthalein excretion in two hours 20 per cent.

Blood —June 3d—three days from onset—the total non

protein nitrogen was 101 mgm for 100 c c., 6/8, Hb , 30 per cent. (Tallqvist), 6/21, erythrocytes, 1,584,000, Hb , 35 per cent. (Tallqvist), leukocytes, 12,600, polymorphonuclears, 82 per cent., small lymphocytes, 19 per cent , large mononuclears, 9 per cent

Blood-pressure 6/2, S P , 135, D P , 80 (auscultatory)
6/8, S P , 140, D P , 80 (auscultatory)
6/10, S P , 120, D P , 55 (auscultatory)

The patient was discharged from the hospital 7/7/16 At that time the urine showed specific gravity 1021, no albumin, no casts Her recovery has been complete

In poisoning by mercury we would expect degeneration of the epithelium of the tubules in its purest form, and you might be surprised to note how the clinical manifestations in this case differ from the one previously described, and from what we have said is characteristic for nephrosis Here we find no marked edema, we note the remarkably prolonged period of anuria, the impaired kidney function, 101 mg of N P N for 100 c.c. of blood, which later would probably have been much higher, the low phenol-sulphonephthalein output, slight manifestations of uremia, and finally the slightly increased blood-pressure Differences in the histologic findings will also be encountered Where in the nephroses cloudy swelling, granular and fatty degeneration were found in the epithelium, especially that lining the proximal convoluted tubules, without marked impairment of kidney function, in the sublimate kidney there is necrosis, cell death, in the same areas with destruction of kidney function There is no evidence of inflammatory reaction in the glomeruli in either It has, therefore, been suggested to designate this as necrotizing nephrosis There is also in the sublimate kidney a tendency to the deposition of calcium in the epithelium, and this, with secondary inflammatory reaction in the connective tissue, is more marked, the more severe and long continued the poisoning There is also found early marked evidences of regenerative processes in the flat epithelium, pyknotic nuclei, and occasional mitoses Edema may occur in the bichlorid kidney, but, as a rule, is absent. The frequent presence of anuria is probably

responsible for the slightly increased blood pressure which occasionally occurs, as well as for the impaired kidney function

CASE III.—B. W., negro, male, aged thirty five. Laborer. Clinical diagnosis, acute diffuse glomerulonephritis plus nephrosis (mixed form).

Anamnesis.—Family history negative. Past history negative, except there was a sore on the penis last year, which was not followed by secondary manifestations of lues. About July 1, 1917, he began to complain of pain in the third finger of the right hand. The pain, which was dull and throbbing in character, grew progressively worse. At the end of a week his doctor made an incision in the finger, on the palmar surface. No pus obtained. The condition of the finger grew rapidly worse. Patient says that the bone "rotted and became unjointed." About this time the entire hand and arm became swollen and he had high fever. About three weeks after the first incision was made the finger was amputated. After this there was gradual improvement. Two or three weeks later patient states that he began to have shortness of breath, that his feet, legs, and abdomen began to swell. This has progressively increased, and on this account he was admitted to Ward E, Memphis General Hospital, September 12, 1917.

Status Præsens.—A well-developed negro man, thirty five years of age. There is marked general edema, including external genitals. The third finger on left hand is absent, amputated at metacarpophalangeal joint. Healing perfect, though the hand is swollen and stiff. Heart apex not located, area of cardiac dulness extends 10 cm. to left of midsternal line. There is a soft systolic murmur at the apex, not transmitted. The pulmonic second sound is slightly accentuated.

Lungs.—Negative, except for the presence of fluid in both pleural cavities, more on the right than the left.

Abdomen.—Ascites—liver nor spleen palpable.

Temperature 97.8° F., pulse, 64, respiration, 24.

Urine.—9/13. Pale, sp. gr. 1010, alb. heavy, casts, pus-cells.

9/25. Pale, sp. gr. 1010, alb. heavy, hyaline and granular casts, no pus.

Urine—10/17 Dark straw, sp gr 1014, alk alb heavy, casts neg
10/18 Dark straw, sp gr 1014, alk alb heavy, casts neg
11/11 Pale, sp gr 1010, alk alb heavy, casts neg

Blood—10/10, Leukocytes, 7400
11/12, Erythrocytes, 4,200,000, leukocytes, 6400, polys, 63 per cent., small lymphs, 34 per cent., large monos, 3 per cent, eosin, 0
Wassermann negative

Blood-pressure 9/21, systolic, 190
10/8, systolic, 172, diastolic, 118
10/16, systolic, 170, diastolic, 115
11/5, systolic, 145, diastolic, 90

Functional Tests

MOSENTHAL KIDNEY TEST-DIET, 11/8/17 DISAPPEARING EDEMA

Time of day	Urine. C.c.	Sp gr	NaCl. Per cent.	NaCl. Gm.	N Per cent.	N Gm.
8–10	280	1010				
10–12	225	1009				
12– 2	215	1011				
2– 4	325	1010				
4– 6	200	1008				
6– 8	250	1012				
Total day	1495		76	11 362	9	13.45
Night	1244	1008	8	9 952	.85	10 57
Total	*2739*			*21 314*		*24.02*
Intake fl., NaCl, and N	1760			8 5		13.4
Difference	979			12 79		10 62

Weight. Kgms	Date	Period	Blood. Mgm urea per 100 c.c.	U	P	T	Urine. Urea gms in 24 hrs	Urea Per cent	
65 1	11/8/17	4–6	48	200		2400	33 6	14	Ambard 09

Phenolsulphonephthalein, 50 per cent. in two hours
CO_2 tension alveolar air, 30 (Marriott)
R p H., 8 2 (Marriott)

This case illustrates the usual etiology of diffuse glomerulonephritis It may be said that all nephritides are due to bacteria or their toxins In the diffuse glomerulonephritis there is usually

a history of streptococcus infection—*e g*, scarlet fever, angina, rheumatic fever, erysipelas, or an infected wound In two or three weeks after the subsidence of the symptoms of the causative infection manifestations of nephritis appear Usually in focal glomerulonephritis the nephritis comes on coincidently with the infection. Here we have an infected wound—after two or three weeks, symptoms of nephritis

As said before, clinically increased blood pressure separates diffuse glomerulonephritis from nephrosis In focal glomerulonephritis there is also an absence of rise of blood pressure, but here the presence of hematuria distinguishes these cases from nephrosis Edema was formerly thought to be one of the distinguishing clinical features of acute nephritis. Many of these cases, however, run their course without ever showing edema at all, and when it is present to a great extent it is, according to Volhard and Fahr, the accompaniment of associated degenerative changes in the epithelium of the uriniferous tubules

The *blood* usually shows evidence of slight anemia, as in this case (reds, 4,200,000), and especially in cases unassociated with nephrosis, hydremia is present.

The *heart* may or may not be hypertrophied It is surprising to see how rapidly the heart enlarges in some cases without edema associated with high blood pressure Even in a few days there may be observed a heaving, displaced apex beat, enlargement of area of cardiac dulness, accentuated second sounds at the base, presystolic gallop rhythm, and systolic murmur at apex

The *urine* usually shows the presence of blood, which gives to it a dark, smoky color Albumin is present, but usually less in amount than in the nephrosis The quantity is diminished and in severe cases anuria may be present. The specific gravity is low in the cases without nephrosis, and possibly the lower the specific gravity, the more severe the injury to the glomeruli The sediment shows red cells, débris, leukocytes, casts, micrococci, and degenerated epithelial cells

The functional capacity of the kidney is usually not markedly impaired According to Volhard and Fahr, the water excreted is good, if no edema is present, as is also the salt excretion The

nitrogen excretion is good The non-protein nitrogen in the blood may be increased and the ability to concentrate present or absent. Marked impairment of function would seem to indicate the presence of severe extracapillary glomerulonephritis with grave prognosis In this case the day and night polyuria and the low, rather fixed specific gravity is probably due to the fact that the patient is eliminating edema The salt and nitrogen excretions are good The urea in the blood and Ambard's coefficient are within normal limits, as is also the phenolsulphonephthalein output in two hours There is evidence of slight acidosis in the alveolar Co_2 tension and R p H

General Symptoms —There is little characteristic in the general symptoms of acute diffuse glomerulonephritis There may be at the onset lassitude, depression, anorexia, increased thirst, severe vomiting, chills and fever, enlarged spleen, or there may be no symptoms at all Changes in the eye-grounds are usually absent, nosebleed and hemorrhages into the skin and mucous membranes may be present. The manifestations of uremia may be divided into those associated with kidney insufficiency, such as contracted pupils, dyspeptic symptoms, great depression, weakness, apathy, stupor, urinous odor of the breath, general hypersensitiveness, muscular irritability, muscular tremors, jerking of the tendons, deep breathing, and fall of temperature, and into those without kidney insufficiency, such as convulsions and symptoms which have been designated eclamptic equivalents, such as amaurosis, hemianopia, transitory disturbances in speech and hearing, paralysis, increased reflexes, positive Babinski's sign, stiffness of the neck, Kernig's sign, headache, and vomiting The uremia with kidney insufficiency rarely occurs in acute diffuse glomerulonephritis, but convulsions are especially liable to occur in children, and they seem to occur independent of the severity of the kidney lesion

Course —This is dependent on the severity of the disease, the time at which it is recognized, and the treatment. With early recognition and proper treatment there is marked tendency to recovery, though many cases pass into the second stage of glomerulonephritis—chronic glomerulonephritis—without kidney in-

sufficiency This patient, who insists on discharge even though he still has an increased blood pressure and albumin in his urine, is already possibly passing into this stage

Pathology —The macroscopic appearance of the kidney in acute diffuse glomerulonephritis shows little that is characteristic, but microscopically the changes in the glomeruli are quite characteristic. Lohlein and Volhard and Fahr lay down as the most important characteristics lengthening and widening of the glomerular loops, absence of blood in the loops and increase of cellular elements in the glomeruli. In the capsular space may be found desquamated epithelium, red cells, coagulated albumin and fibrin, the walls of the capsule may be adherent. In the tubules are coagulated masses composed of red cells, white cells, and coagulated albumin forming casts

In isolated proximal convoluted tubules may be found granular degeneration of the epithelium, Henle's loops, and the collecting tubules may contain the above-described casts The extent of the changes in the tubules depend upon the extent of the inflammatory process in the glomeruli to which they belong Extensive degenerative changes in the tubules are present only in the cases of mixed form—nephritis plus nephrosis

With severe changes in the glomeruli small-cell infiltration in the connective tissue may be present. There are no changes in the blood vessels

Case IV —Mr ———, white, aged forty two, merchant. Clinical diagnosis, end stage chronic glomerulonephritis—secondary contracted kidney

Anamnesis —Family history negative. He had the ordinary diseases of childhood, but never had scarlet fever or diphtheria. As a youth he had a good deal of sore throat. He has considered himself very healthy until about three months ago, when he began to have shortness of breath, and he consulted me at this time, complaining of shortness of breath, swelling of the feet and ankles, and impaired vision

Status Præsens.—A well-developed, intelligent gentleman, 6 feet in height, weight 220 pounds and forty two years of age The skin and mucous membranes are pale The feet and legs

are swollen and pit on pressure He is dyspneic The lungs are negative except for faint râles at the base posteriorly The apex-beat is displaced downward and outward, the area of cardiac dulness is increased to the left and right, there is a faint systolic murmur at the apex, transmitted to the axilla, the second sounds at the base are accentuated, orthodiagram 6 5, 11 5, 19 5 cm , radial pulses equal 90 per minute, pulsus alternans, radials not palpable

Abdomen —No ascites, liver palpable two fingerbreadths below the costal margin and tender, spleen not palpable

Blood-pressure —S P , 170, D P , 100 (auscultatory)

Blood —Red cells, 2,640,000, Hb , 32 per cent. (Sahli), C I , 6, leukocytes, 13,000, polys , 84, S Ls , 8, L Ms , 8, Eos , 0

Urine —Single specimen, pale, sp gr 1005, faint trace of albumin, no sugar, casts present, leukocytes and red cells present.

Eye-grounds —Normal Impaired vision due to error in refraction (Dr Farrington)

Functional Tests

MOSENTHAL DIET

Time of day	Urine. C.c.	Sp. gr	NaCl. Per cent.	NaCl. Gm	N Per cent.	N Gm.
8–10	200	1005				
10–12	250	1005				
12– 2	250	1005				
2– 4	250	1005				
4– 6	200	1005				
6– 8	160	1005	.45	5.87	25	4 375
Total day	1310					
Night 8–8	1750	1005	3	5 25	288	3 772
				11 12		8 147
Total twenty-four hours	3060			8.5		13
Intake fl , NaCl, and N	1760			2 62		5 267
Difference	1300					

Urea per 100 c.c. of blood, 324 mgm. Phenolsulphonephthalein in two hours, o.

Under rest in bed, a low protein diet, and measures directed to the heart this patient improved for about two weeks, but within six weeks from the time of examination he died in uremia

This is a rather typical case of the end-stage of chronic glomerulonephritis—the so-called secondary contracted kidney Often, as here, we are unable to discover the cause of the condi-

tion It is, however, always the development of the previous stages This development may be rapid, requiring only a short time, or extend over a period of years The previous stages of the disease may be latent so far as symptoms are concerned, and the first manifestations show themselves in the fatal kidney in sufficiency associated with cardiac insufficiency, as this case also illustrates More frequently, however, they run a course of more or less chronic or periodic manifestations

The increase of blood pressure in these cases is usually marked and is probably more constant the older the process

The heart is usually hypertrophied, but, as a rule, not to the same extent as in the scleroses There is a marked tendency to myocardial insufficiency, as seen in the dyspnea, presystolic gallop rhythm, increase in area of cardiac dulness, swelling of the liver, and edema of the lower extremities

Edema of the nephritic type is seldom seen unless the nephritic process is associated with marked degenerative changes in the tubules The urine is pale, different specimens showing a remarkably constant color, increased in amount, albumin is present in small amount, and the sediment shows leukocytes, casts, degenerated epithelium, and detritus

Renal insufficiency is the chief clinical characteristic of this type of nephropathy As seen in this case, there is a remarkably low fixed specific gravity, a night associated with a day polyuria, low night specific gravity, low salt and nitrogen concentration, a high urea content in the blood and a low sulphonephthalein output. According to Stengel, Austin, and Jonas there is also in these cases a low plasmachlorid content, as also a reduction in the plasma bicarbonates

General Symptoms.—There is usually great loss in strength. The patient often presents a cachectic appearance The skin and mucous membranes are pale, the hair dry, with a tendency to fall out, frequently there are hemorrhages into the skin and mucous membranes, conjunctivæ, epistaxis, menorrhagia. The blood shows a marked tendency to anemia in marked contrast to benign sclerosis

Changes in the eye-grounds are frequent, hemorrhages as

well as true albuminuric retinitis being present The most frequent termination is in that type of uremia associated with kidney insufficiency, though convulsions and eclamptic equivalents may occur with cases associated with arteriosclerosis, which is not infrequent

As said above, the course is variable, but the outcome is always the same—death

Pathology—The kidneys in these cases are pale and usually contracted, but the size varies greatly Microscopically there are the evidences of glomerulonephritis with secondary fibrosis Due to the repeated injury which the kidneys have sustained, the damage to the glomeruli with atrophy of the tubules belonging to them, and regenerative changes in the remaining portions, there is quite a variegated picture Very frequently there is an accompanying sclerosis of the blood-vessels

CASE V—Mr ———, white, aged seventy-four Planter Clinical diagnosis, benign hypertension

Anamnesis—Famly history negative Has never had any serious illness Is a large planter and has had much worry For the past two years has noticed he had shortness of breath on exercise Lately he has been worried a great deal with cough and bronchitis Occasionally his ankles swell during the day, but the swelling usually disappears after a night's rest His doctor tells him that he has a lazy heart

Status Præsens—A gentleman in excellent condition of nutrition and does not look as old as he really is His voice is hoarse and he has a slight cough His eyes show a distinct arcus senilis, the ankles are slightly edematous His radials are palpable, the pulses equal and 84 per minute There is an occasional extrasystole The area of cardiac dulness is increased to the left Orthodiagram 4 5, 12 5, 19 5 cm The heart sounds are clear, the aortic second is accentuated No murmurs present

There are râles throughout both lungs The liver is palpable two fingerbreadths below the costal margin The spleen is not palpable The abdomen otherwise negative Nervous system negative Temperature, 98⅘° F

Blood-pressure—S P , 195, D P , 95

Blood —Erythrocytes, 5,130,000, Hb , 90 per cent. (Sahli), leukocytes, 8400, polys , 74, Sls , 20 per cent., L Ms , 5 per cent., Eos , 1 per cent

Functional Tests

MOSENTHAL'S KIDNEY TEST-MEAL

Time of day	Urine. C.c.	Sp. gr	NaCl. Per cent.	NaCl. Gm.	N Per cent.	N Gm.
8–10	120	1015				
10–12	155	1013				
12– 2	115	1014				
2– 4	190	1008				
4– 6	200	1006				
6– 8	105	1010				
Total Day	885		7	6 195	.8	7.08
Night 8–8	455	1010	.8	3.64	9	4.095
Total twenty-four hours	1340			9.835		11 175
Intake	1760			8.5		13.4
Difference	420			1 335		1 225

Phenolsulphonephthalein in two hours, 50 per cent.

Benign hypertension usually occurs in the advanced periods of life, a hereditary tendency is frequently noted, but the real cause of the condition is unknown

The blood pressure is high, and when the disease is well established is constantly so, but in the early stages may show marked variation. The symptoms are more cardiovascular than renal. The heart is markedly hypertrophied, which at first involves the left heart, giving fluoroscopically the so-called sock heart. With relative insufficiency of the heart muscle there is a feeling of oppression in the chest and dyspnea on exercise. Disturbances of sleep are frequent, pounding noises in the ears when the patient lies on the left side, disturbing dreams, nocturnal attacks of dyspnea prevent sleep The aortic second sound is accentuated, and a presystolic gallop rhythm is frequently present With greater dilatation signs of decompensation occur Stasis bronchitis, pulsation in the veins of the neck, systolic murmur at the apex, swelling of the feet and swelling of the liver Extrasystoles, pulsus alternans, tachycardia, and heart block may occur

Edema is lacking in the compensated cases There are

numerous symptoms on the part of the vascular system—*e g*, cerebral hemorrhage—various symptoms on the part of the nervous system due to cerebral vascular changes, Cheyne-Stokes' breathing, etc

There are few symptoms referred to the renal system There are only slight changes in the urine except those referred to the failing heart The functional capacity of the kidneys is little impaired as a rule The blood count is usually normal In the eye-ground there is frequently evidence of arteriosclerosis, but never true albuminuric retinitis Manifestations of true uremia seldom occur

The course is essentially chronic, and with proper régime the patient's life may be prolonged for an indefinite period The end usually comes through cardiac insufficiency or vascular accident

The kidneys may or may not be contracted There are arteriosclerotic processes in both the large and small vessels, but no evindece of inflammatory changes in the glomeruli.

CASE VI —N W, male, negro, aged sixty-seven Laborer Clinical diagnosis, arteriosclerosis and nephritis—combination form

Anamnesis —Family history negative Had typhoid at thirteen years of age Pneumonia at about the same time Smallpox at fifty years of age Had an arthritis of right ankle then, and of the left five years ago Had fever and was confined to bed four or five weeks His present trouble began October 3, 1917, with pain in the right side of the abdomen, shortness of breath, swelling of feet and legs, and hiccup On account of these symptoms he came to the hospital and was admitted to Ward E, Memphis General Hospital, October 22, 1917

Status Præsens —A negro man in rather a poor state of nutrition, mucous membrane pale He is dyspneic, sitting up to enable him to breathe better, complains of pain in his abdomen, and has hiccup

Lungs —Dulness and diminished breath sounds over both bases, râles irregularly distributed over both lungs

Heart —Apex located in the sixth interspace, 12 cm to the

left of the midsternal line, right border of cardiac dulness extends 4 cm to the right of the midsternal line. Supracardiac area of dulness increased There is a systolic murmur at the apex, transmitted to the axilla The aortic second is accentuated and there is a systolic murmur in the area.

Abdomen —Tender, no rigidity, wall edematous, liver palpable, spleen not palpable

Extremities —Feet and legs edematous

Eye grounds —Not examined

Blood —Erythrocytes, 4,428,000 Whites, 8400 Polymorphonuclears, 69 per cent., small lymphocytes, 28 per cent., large mononuclears, 3 per cent. Wassermann positive (++++)

Blood-pressure —Systolic, 170, diastolic, 100

Urine —Sp gr 1015 Albumin, casts

Functional Tests

MOSENTHAL DIET, OCTOBER 24, 1917

Time of day	Urine. C.c.	Sp. gr.	NaCl. Per cent.	NaCl. Gm.	N Per cent.	N Gm.
8–10	74	1014				
10–12	115	1010				
12– 2	102	1015				
2– 4	124	1015				
4– 6	200	1010				
6– 8	380	1010				
Total day	995		.4	3.988	18	1 704
Night, 8–8	410	1010	.3	1.23	.32	1 75
Total day 24 hours	1405			5 218		2.934
Intake fl., NaCl and N	1760			8.5		13.4
Difference	353			3 282		9.466

Weight.	Period.	Blood. Mgm. U Per 100 c.c.	Amt. U P	Total	Urine. Qnt. Urea in 24 hours	Urine Per cent. 19 5	Ambard coeff
65.4	10–12	180	115 c.c.	1380	26.91		.357

Alveolar CO_2 tension 25 (Marriott) alkali reserve 7.8 (Marriott), phenol sulphonephthalein 20 per cent. in two hours.

The patient of his own accord left the hospital 11/11/17 improved

In the combination form there are the symptoms described above plus those due to the associated nephritis, which depend

upon the extent of the nephritic process The patient begins to lose weight, the skin to assume the characteristic nephritic appearance, albuminuric retinitis is frequent, and symptoms due to kidney insufficiency develop

The course is variable and death occurs either from myocardial insufficiency, vascular accident, or uremia

From these selected cases we believe that it is often possible to classify the nephropathies clinically as well as pathologically on a degenerative, inflammatory, and sclerotic basis But it must be remembered that transition forms and combinations frequently occur which render an exact classification difficult. It, however, has many advantages from a diagnostic, prognostic, and therapeutic standpoint

CLINIC OF DR. GEORGE S BEL

CHARITY HOSPITAL, NEW ORLEANS, LA

A COMPARISON OF THE ESSENTIAL PNEUMONIAS FROM THE STANDPOINT OF THEIR CLINICAL SIGNIFICANCE

ANATOMICALLY, we recognize two forms of inflammation of the lungs, which are determined according to whether the pathologic process has to do primarily with the terminal air sacs of a lobe or with a bronchus It is essential in every pneumonia to determine which one of these processes we are dealing with, since it has a direct bearing on the etiology, and thus subsequently on the treatment. It is well recognized nowadays that the type of pneumonia called the lobar or croupous is only caused by some one of the strains of the pneumococcus, while the other form of pneumonia has a variety of exciting agents In this connection bronchopneumonia is a type of lesion always determined by tuberculous, influenzal, streptococcal, typhoidal, meningococcal, and bubonic infections of the lungs In my experience the most of these bronchopneumonias are secondary infections of the lungs In other words, bronchopneumonia usually follows or is superimposed upon some other infection With the exception of the pneumococcal infection of the pulmonic tissue the injury invariably begins in connection with the terminal bronchial twigs The pneumococcal infection, on the other hand, never shows the focus of injury in the bronchial tree Furthermore, no injury whatsoever can be demonstrated, though it is thought that possibly the lining epithelial cells of the air-sacs are injured in this form of pneumonia

Lobar pneumonia which is produced by one of the several variants of a pneumococcus species invariably has a sudden abrupt onset, with more or less fever, chill, pain, and symptoms

of toxemia The lung is found to have involved at least a lobe In other words, there is a large focus of infection, which is in marked contrast to a small focus or foci, so characteristic of the other form of pneumonia A physical examination early in this infection reveals that a large portion of the lung is involved, which is never the case in the early part of the broncho type of the infection Of course, it must be recognized that bronchopneumonia that is rapidly advancing and has been under way for some time may give you the same amount of lung involvment as seen in lobar pneumonia It is this very point that I wish to stress, since I believe that too often a bronchopneumonia is mistaken by the clinician by the physical signs for the lobar type This is especially true of the so-called deep-seated central lobar pneumonias, which, in reality, are broncho in type, borne out by careful and painstaking autopsy study Furthermore, it is a matter of clinical as well as pathologic knowledge that lobar pneumonia is a simultaneous involvement of a whole lobe or lobes, while in bronchopneumonia this is never the case On the contrary, the bronchopneumonia begins insiduously as a deep-seated bronchitis with subsequently the slow development of small contiguous areas of consolidation There may be and there often are multiple foci of infection in bronchopneumonia, which in part account for the patchy nature of the consolidation, while in lobar pneumonia there is always one large focus

As to the inflammation, there is no essential difference in the exudate of either pneumonia They are both acute, and may be characterized by serous, purulent, or fibrinous accumulations There is this difference, however, that in the one, namely, lobar the exudate is confined to the terminals of the respiratory tree it having begun and ended here, in bronchopneumonia the exudate is not only in the terminal air-sacs of one or more systems, but it is in the interstitial tissues, such as the peribronchia and interalveolar structures, and in every instance begins in the lining of the bronchial tree The areas of consolidation are secondary to the primary bronchitis, having developed subsequently as an extension from the original focus In this fact we have an explanation of the development from time to time i

the course of bronchopneumonia of new isolated foci of consolidation In other words, a new crop of lesions may appear from time to time during the course of infection These new foci are always at first small and scarcely to be detected, but if carefully followed will be found to gradually increase in size. As time goes on these bronchopneumonic foci may attain such a size as to meet or coalesce, giving us practically a whole lobe involvement. If the case is seen at this period of the disease it is readily understood how the clinician may wrongly call it one of lobar infection However, if you bear in mind what I just said you may avoid confusing the bronchopneumonia with the lobar pneumonia, as in the former, even though the whole lobe is practically solid, you will detect on careful physical examination a variety of signs indicating that the inflammatory process is of various stages, though we do not recognize in bronchopneumonia the gross pathologic stages of lobar pneumonia, such as engorgement, red and gray hepatization.

I wish to emphasize that it is well to remember in uncomplicated lobar pneumonia the lung tissue in convalescence is returning to normal, while in bronchopneumonia the lung, at best, is only partially normalized. This is because in the one the inflammatory process did not involve the supporting tissue of the lungs, and in consequence resolves without organization, while in the broncho type the inflammatory process involves the supporting tissues and, of necessity, is organized during repair To state in other words, recovery after lobar pneumonia leaves the lung, in an uncomplicated case, anatomically and physiologically as it was before the infection, in bronchopneumonia the lung is in these respects invariably impaired The importance of this is obvious For example, a lung thus permanently impaired, even though slightly, renders it vulnerable to other infections, more especially tuberculosis In children this is particularly the case where pulmonary tuberculosis develops subsequently into a bronchopneumonia

Now gentlemen, I shall bring before you cases which illustrate what I have had to say about the essential pneumonias We will now study and discuss together these cases by carefully comparing

the clinical symptoms and physical signs so we can intelligently interpret the pathologic processes which underlie these essential pneumonias

This case, negro male, age thirty-one, occupation laborer, admitted to my service March 15, 1918, previous history unimportant, as it has no bearing on his present illness

Present Illness—Patient taken suddenly sick four days ago, while at work, with classical symptoms of lobar pneumonia.

At this bedside examination we notice the following symptoms and signs Temperature 104⅘° F, which has oscillated between 103° and 104⅘° F since his admission Pulse 130, varying between 118 and 124, of fair volume and regular Respiration 44, slight delirium, herpes on lips, and, as you notice, the patient grunts on expiration This individual is coughing and expectorating sputum which is tenacious and blood stained Blood-picture in this case shows a leukocytosis of 26,000, the culture reveals the pneumococcus, which not only agglutinates with the patient's blood, but with a monovalent serum, classifying it as strain No 3 Blood-pressure at this time is 118 systolic. The urine, as you notice, is one of any acute infectious disease.

The following physical signs are present

Respiratory movements and expansion are much diminished on the right side, tactile fremitus is greatly increased and absolute dulness over the whole right lower lobe and marked bronchial breathing, with bronchophony

The second case is one of bronchopneumonia, of influenzal etiology This case, a negro male, aged eighteen, occupation laborer, admitted to my service on March 10, 1918, with a history of having a severe coryza, cough, slight fever, and indefinite pains over various parts of the body As in the other case, the patient's past history is of no moment

Now, gentlemen, what are the clinical symptoms and physical signs to be found in this case? We will determine these and then compare them with those of the lobar case

Let me call your attention to the fact that the onset of the lung infection was secondary to the infection already existing in the upper respiratory tract and that it began insidiously, develop-

ing gradually without a chill or high fever and very little additional signs and symptoms to those present prior to the lung involvement, which is in contrast to our other case there of lobar pneumonia We have said that this bronchopneumonia is influenzal. The statement is made on the laboratory findings of Pfeiffer's bacillus in large numbers in the sputum The agglutination reaction of the patient's blood is also positive While we know the influenza bacillus is responsible for the bronchopneumonia, it may not be entirely so We must bear in mind that this and other bronchopneumonias have two or more causal agents working conjointly We call this case influenzal bronchopneumonia because there are laboratory reasons pointing to the fact that the Pfeiffer organism is playing the most important rôle.

At this examination we notice the following symptoms His temperature is 103° F and is of an irregular type, in that, as we see by the chart, there is a range of 3 degrees within a twenty-four hour period, which was not like that in our chart of the lobar patient. The cough is persistent, frequent, and of a dry character, also differing in this respect from the cough of the lobar patient. It is a significant fact that in this patient there is no acute pain in the chest like we have in our other case, which indicates the inflammatory process does not involve the pleura so typical of the lobar type This patient has dyspnea and rapid pulse that does not differ from the other pneumonia, and the explanation is the same The blood picture in this case is interesting While we have a leukocytosis, it is slight compared with the one we have in the other patient. The increased neutrophils are due to the mixed infection, because pure influenzal infection would give us a leukopenia Therefore the leukocytosis here is further proof that the bronchopneumonia is caused by two or more etiologic factors

The chief physical signs are indicative of isolated areas of consolidation intimately associated with bronchitis While we cannot detect here from this single examination that the solidifications are increasing in size and new ones developing, we can tell you, however, that such is the case Day before yesterday I

noticed that several of these pneumonic patches found today were much smaller and that we have others that were not present before

To appreciate the pathology of bronchopneumonia one must go over the physical signs from day to day, for in no other way can you perceive that the inflammation is spreading insidiously by continuity and contiguity All of this is in marked contrast to the development of the process of our lobar pneumonia case We notice scattered areas of impaired resonance, with alternating hyperresonance, also detect subcrepitant râles occasionally, but the predominating râle is the pipling dry type which outsounds the other in loudness This kaleidoscopic change is only appreciated by an understanding of the pathology Just as we have changes from day to day in the size of the consolidation, so have we changes in the consistence of these areas which explains why an area resonant yesterday may be dull today and why these râles also vary in position and in character The loud wheezing râles in bronchopneumonia are due to the inflammation of the bronchioles, which is a primary and continuous process of this disease

Now gentlemen, to explain these symptoms and signs we must bear in mind the anatomy, physiology, and pathology of the organs involved While we here are concerned mainly with the pathology, we cannot understand this unless we know the anatomy and physiology, the three are essential if you are to appreciate the signs and symptoms in this case Students of medicine must have an explanation of the various manifestations on the part of the host who is combating an injurious agent if they are to intelligently diagnose and subsequently treat the disease Too often the student learns a group of signs and symptoms for a given infection without understanding their mechanism of production, and for this reason I will now proceed to elucidate the symptom-complex that we have here before us occasioned by the pneumococcus infection of the lungs

The temperature is occasioned by what? In a general way we say that all infectious diseases give us increased body heat, which is true, but does not explain its mechanism of production

We believe it is the result of the direct action upon the body cells by the poison elaborated by the pneumococcus This action of the poison upon the cells increases their activity and thereby causes more heat production There is also a direct stimulation by the poison upon the heat producing centers which is not commensurate with heat dissipation We said the pulse rate or heart action and respiration are increased in this case Why? The explanation of the cause is rather complex Three factors at least are concerned In general, we may say the specific poison is directly or indirectly responsible The poison produces pyrexia in the manner previously mentioned, and this fever accelerates the heart because of the increased body heat. Second, the poison is thought to have a depressing effect upon the inhibitory cardiac fibers of the vagus, which permits, in consequence, an acceleration of the heart action Third, and I think most important, is the direct action upon the parenchyma of the heart by the poison which brings about weakening of the heart muscle because of the degeneration This lowers the blood pressure, and the heart, to compensate, beats faster Furthermore, the lowering of the blood pressure means less oxygenation. There is one thing I wish to call your special attention to, and that is, that the consolidation of the lung is in no way responsible for the increased heart action, the dilatation of the right chambers, or the dyspnea If the solid lung was the cause of these symptoms, how do we explain the rapid return to normal of the heart, its action, and the dyspnea immediately following the crisis, when we know that there are several days during the resolution stage in which the lung is still solid, as demonstrated upon physical examination? The *herpes* is thought to be the result of irritation and stimulation of some one of the posterior root ganglia The *pain* which is so common and constant with lobar pneumonia is due to the inflammatory process involving the pleura The pain is the expression of irritation of the nerve-endings by the toxin, more so, we think, than we formerly thought to the pressure or stretching of the nerve-endings by the surrounding exudate In this connection let me impress you with the fact that lobar pneumonia is always associated with pleurisy, differing in

this respect from the broncho type, except in those cases, and as a rule, few in number, where the inflammatory process begins at the periphery or extends to the periphery It may be said that no bronchopneumonia begins with an acute pleurisy, because even with those cases where the focus began at the outer margin the inflammation was not exactly in the pleural surface, but some distance below Regarding the sputum in the early stage, it is likely to be scant and of a mucoid nature Later it becomes more profuse, blood streaked, or rusty colored In bronchopneumonia there is always more expectoration in the disease and its character is mucopurulent, and not so likely to be blood stained, and rarely rusty This difference in the sputa in the two pneumonias is readily explained by the pathology In lobar pneumonia the sputum is composed largely of the hypersecretion from the respiratory tubes, while in bronchopneumonia it is the actual inflammatory exudate mixed with the hypersecretion The sputum in lobar pneumonia is thick and tenacious because of the mucus predomination, and later on in the disease when it becomes creamy it loses its tenacity because of the preponderance of inflammatory exudate which has undergone more or less digestion The rusty color of the sputum is due to the blood which has hemolized The blood-picture in lobar pneumonia does not differ materially from bronchopneumonia or, for that matter, from that of any other pyogenic infection It is the rule to have a very high neutrophilic leukocytosis in lobar pneumonia for the reason that there is a tremendous focus of acute inflammation which drains a large vascular area of the essential cells of acute inflammation Without exception lobar pneumonia is the largest acute inflammatory area that we know for any infection In explanation of the occurrence of leukocytosis we believe that it is the replenishing of the normal neutrophilic element to the circulation by the blood-making tissue in lieu of the loss occasioned by the tissues inflamed The local attraction or positive chemiotaxis of the leukocyte is enormous in lobar pneumonia because of the size of the lung area involved, together with the fact that there is a large number of blood-vessels that are normally present of necessity in the lung The drain on the vascular system

through the vessels involved is so large there must be a compensatory replenishment of the neutrophils to the circulation. It might be asked, Why the need on the part of the host for an increase above the normal leukocyte in the circulation during lobar pneumonia infection? I believe there is no need, but in accordance with Weigert's law of superproduction we have the true explanation of the circulatory leukocytosis The blood making tissues are called upon to make up the loss in neutrophils which have been attracted from the vessels to the inflammatory focus, and in making up the loss the tissues overdo it, just as we have in the case of repair to an injury or the production of antibodies It is well to remember occasionally we may have in lobar pneumonia a low leukocytosis or even a leukopenia, which signifies in both instances a low-grade resistance on the part of the patient and necessarily a poor prognosis

In explanation of certain physical signs presented in this case I wish to say they are, in general, brought about by the non functionating solid lung tissue. The lagging or immobility of the affected side is the result of pain from the pleuritis and the consolidative pulmonic tissue The other physical signs, such as increased tactile fremitus, dulness, bronchial breathing, and bronchophony, are due to the solidification of the lung tissue The consolidation is due to the obliteration of the normal air spaces by exudate, which has eliminated the air The exudate is derived almost entirely from the interalveolar capillaries, with the exception of the fibrin and desquamated epithelial cells of the air-sacs The fibrin, one of the main constituents of the exudate, does not appear early in the pneumonia because it is derived later from the elements of the exudate which has already arrived Fibrin, unlike the neutrophils, serum, and red blood cells, is subsequently formed in the air sac spaces, the result of fibrinogen and lime salts of the serum and nucleoprotein or ferment from the disintegrated neutrophils The greatest degree of solidity in lobar pneumonia is when the maximum of fibrin has been obtained. The lung at this period is not only solid but granular gray, and dry The degrees of consolidation are explained by the character of the exudate Early in the disease the exudate is

chiefly serous and does not take up the entire air spaces, which gives you a less firm consistence, together with considerable sponginess because of the contained air not yet excluded, later in the disease the exudate becomes less fluid and more solid because of the appearance now of more cells and fibrin and the complete exclusion of air It must be remembered that after the crisis the lung again approaches the original consistence by passing from the greatest degree of firm solidity to a semifluid and then a fluid, and finally an air-containing fluid consistency, indicating that the air is again returning

While I have not attempted to describe the entire symptom-complex of this case of lobar pneumonia, I have taken up what I think are the essentials

Lobar pneumonia terminates in resolution with a complete return of the lungs to normal, or in partial resolution with more or less organization, resulting in permanent impairment or fibrosis of the lung after recovery Complete resolution is the more common of the two favorable endings, and is, of course, to be desired It is of interest to know that in uncomplicated pneumonia with recovery we have the only example of an extensive inflammatory exudate being removed without organization The termination in the ordinary case in this infection is what we commonly term "crisis," that is, a sudden sharp fall in temperature to normal. The cause of the crisis is the neutralization of the pneumococcal toxin by the antisubstances that have now been developed by the host in sufficient quantity Pneumonia when uncomplicated is a self-limited disease with a sudden onset, profound toxemia, and abrupt termination, after a serious illness of about from seven to nine days

The **treatment of lobar pneumonia,** aside from supporting, should be serum therapy Regarding the treatment other than serum the principal object is to support the patient in every way possible, to augment his powers to resist the inroads of the infection In other words, to meet the indications as they arise, bearing in mind that drugs have no specific action and are to be avoided as much as possible It hardly seems necessary for me to mention that fresh air, nutritious diet and stimulation, when

indicated, should be administered It is essential in this connection to determine as early as possible the type of pneumococcus giving rise to the pneumonia I do not advocate determining the type from the sputum or upper respiratory tract when we have a much shorter and more satisfactory route All pneumococcal pneumonias are septicemias, particularly is this true in the early stages of the disease. To work out the strain from the sputum means first a separation from the upper respiratory flora, as well as the separation possibly from other strains of the pneumococcus This is time-consuming, whether you use the cultural or animal method of differentiation. On the other hand, a culture from the blood gives you the organism pure, which can be immediately tested against the various immune sera to determine the type. Even in the event of failure to recover the organism from the blood-stream, stock pneumococci, representing the four strains, can be tested against the patient's blood, which in my experience contains sufficient agglutinins as early as the second day of the disease to diagnose the type of the infection Three days at least are required to determine the pneumococcus strain when the sputum is employed As to the value of the various types of pneumococcal antitoxins, we infer from Cole, of the Rockefeller Institute, that the serum for type 1 is highly efficacious, while for 2, 3, and 4 its value is questionable.

While speaking of specific treatment I would like to state that vaccine therapy, whether the plain or sensitized product is administered, has not in the experience of most clinicians yielded results In my opinion, vaccines for the cure of pneumococcal pneumonia has failed completely I strongly advise the serum therapy for lobar pneumonia because it undoubtedly is beneficial in certain types of the infection and may be of considerable value in other types Unlike vaccine therapy, serum cannot be harmful in any case. There is always the question of overburdening the immunity mechanism by the introduction of more poison, which one is invariably doing when he injects specific vaccines. Vaccines, I believe, we may say unreservedly are more likely to be beneficial in localized than in generalized infections Lobar pneumonia, in the strict sense, is not a localized infection.

I have indicated to you from the beginning of this lecture, the disease is a bacteriemia, producing changes toxic and otherwise in various parts of the body than the lung

We hope and we have good reason to believe from the advance in serum therapy made by Dr Cole that the time is not far distant when we will have a specific treatment for lobar pneumonia as efficacious as we have for diphtheria.

The treatment of bronchopneumonia is based largely upon the determination of the injurious agent, that is, as far as specific treatment is concerned, and in the experience of all, this is decidedly limited in its effect Theoretically, vaccine or serum therapy is advocated, but practically it has not yielded results So many of the bronchopneumonias are secondary infections and localized, that vaccines suggest themselves for treatment. As a preventive measure against the development of this type of pneumonia vaccines might be advocated, for example, influenzal infection primarily in the upper respiratory tract if administered at this time may prevent the subsequent development of bronchopneumonia As far as non-specific treatment is concerned, what has already been said about the lobar type applies to the bronchopneumonia

TYPHOID FEVER COMPLICATED WITH PURULENT CEREBROSPINAL TYPHOIDAL MENINGITIS AND NO INTESTINAL LESIONS

THE case which we have for conference today is one which has been under our observation in the ward as typhoid fever with meningitis The object of this conference is to establish from the postmortem findings to what extent we were correct in our clinical diagnosis This case is one of unusual interest because of the cerebrospinal inflammation, which has been prominent since three days after his admission to the ward seven days ago Shortly after admission the meningeal symptoms became so marked in this case of typhoid fever we suspected metastasis to the brain or a superimposed infection due to some other micro-organism. To clear up this point a lumbar puncture was made and a very turbid fluid removed which, on microscopic examination, revealed the Bacillus typhosus Prior to the meningeal involvement the case presented many of the clinical features of typhoid fever, which was confirmed by the laboratory through a positive blood-culture and a positive Widal reaction.

Purulent meningitis due to the Bacillus typhosus is exceedingly rare as a primary infection and not at all common as a complication in typhoid fever I do not wish you to confuse actual infection of the meninges by the typhoid bacillus with the clinical meningismus, which is not at all uncommon in typhoid patients

Now, gentlemen, before we begin the autopsy, we will review the bedside findings together with the history of the case.

J K., white, male, aged twenty years, occupation laborer, native of Louisiana, admitted to my service in the Charity Hospital April 2, 1918

Previous History —Had the usual diseases of childhood, otherwise unimportant

Present Illness—About eight days ago began to complain of slight frontal headache, general malaise, and on one occasion had a slight chill On admission he presented the following symptoms and signs Headache, fever (102⅘° F), pulse 96, respiration 26, tongue coated with red edges Lungs and heart were negative Abdomen was distended and sensitive. Spleen and liver palpable There were no rose-colored spots to be detected, white blood-cells 7000, with 64 neutrophils, 36 lymphocytes Widal positive At this time a blood-culture was requested and forty-eight hours later reported upon as positive to typhoid fever

The signs of meningeal disease appeared three days after admission and a lumbar puncture was made at once The spinal fluid was under pressure and turbid A pure culture of the typhoid bacillus was recovered From this date the patient presented the classical symptoms of meningitis, such as rigidity of neck, divergent strabismus, absence of patella reflex, and positive Kernig Twenty-four hours before death patient became convulsive and comatose

I shall not take up too much time with the explanation and description of the symptomatology that was presented in this case because we are anxious to see the pathology After the postmortem, which is to be held immediately by my colleague, Professor Duval, we will together discuss this case from the pathologic and clinical aspects I wish to impress the class with the importance of postmortem study of all cases, and particularly cases that have been followed by the students in the wards There is no better way of learning your medicine than in conference at the autopsy table

Postmortem Examination.—*Summary of the Protocol*—The body is that of a well-developed and nourished white male, length 160 cm Rigor mortis is marked, lividity is noted in the depending parts of the body There is no edema The pupils are equal and measure 4 mm

Peritoneal cavity contains about 100 c c of a reddish-colored fluid The omentum is short and curled up about the transverse colon The mesenteric lymph-nodes are enlarged throughout and

on section are distinctly meaty The appendix is negative and measures 15 cm in length The diaphragm reaches the fifth interspace on the left and fifth rib on the right

Pleural cavities contain a slight excess of clear fluid, otherwise negative

Pericardial cavity is negative.

Heart weighs 330 grams, but otherwise normal, except that it is somewhat friable The endocardium and valves are negative

Lungs are negative

Spleen weighs 290 grams, is enlarged, and rather soft. The malpighian bodies are obscured The splenic pulp is very soft, chocolate brown in color, and readily comes away on scraping

Liver weighs 2020 grams On section the cut surface presents many yellowish areas, ranging in size from a dime to a silver dollar The gall bladder and ducts are patent.

Pancreas is negative

Gastro-intestinal tract is absolutely negative throughout. In other words, there is no evidence of ulceration of any description Peyer's patches and solitary follicles are normal.

Adrenals are normal

Kidneys weigh 650 grams The capsule is tense and, on section, the cut surface presents a swollen, cloudy appearance. The malpighian tufts are enlarged and distinct as elevated tiny red dots. The cortex measures 8 mm in greatest depth

The organs of the neck are negative.

Genital organs are negative.

Head covered with short, light brown hair Scalp and calvarium are normal Dura non adherent. Villi numerous Brain weighs 1400 grams On opening the dura the pia and arachnoid are markedly injected, and there can be noticed beneath that the brain is bathed in a purulent exudate The convolutions are flattened and the sulci are correspondingly obliterated This exudate is found most marked over the cortex, though it is present everywhere beneath the covering of the brain and spinal cord On section of the brain, the ventricles contain a slight excess of fluid which is turbid The nasal ganglia, pons, and medulla show nothing remarkable.

Cultures from the heart's blood and exudate of the meninges subsequently revealed a pure growth of Bacillus typhosus

Anatomic Diagnosis—Typhoid fever, without intestinal lesions, typhoid cerebrospinal meningitis, typhoid septicemia

From the anatomic findings we notice that the gross lesions in the spleen, the liver, the lymph-nodes, and the kidneys are those of classical typhoid fever, which bears out our clinical diagnosis in this case The most interesting features of the autopsy are the absence of typhoid lesions in the intestinal tract, and the presence of purulent typhoid cerebrospinal meningitis Both are unusual To explain the absence of any intestinal lesion we might say that the case, because of the meningitis, died before they could develop, or, on the other hand, that the intestinal lesions, because of the long duration of the disease, had healed Against the first hypothesis is the normal condition of Peyer's patches and solitary follicles Cases of typhoid dying from toxemia in the intestinal pre-ulcerative stage always show a more or less swollen condition of Peyer's patches Even where it is too slight to be detected in the gross, there is abundant microscopic evidence which is unmistakable On the other hand, the intestinal lesions, had they existed, and had healed, would show evidence in the Peyer's patches of either scarring or pin-point areas of altered hemorrhage We can assume from the facts in hand that no intestinal lesions of typhoid existed in this case

Turning to the meningitis, we are not only struck with its extent, but with the character of the inflammatory exudate. The typhoid bacillus is ordinarily not a pus producer, but excites a proliferative reaction on the part of the host. When, however, it plays the rôle of a pyogen it is extremely interesting because unusual In this case we have positive evidence of the organism playing a twofold rôle, namely, exciting proliferation in one place and exudation in another This condition is so unusual for the typhoid organism that we are obliged to look with suspicion upon the purulent exudate as of typhoidal origin until all other organisms are bacteriologically exhausted, which has been done in this instance

It used to be held by some good authorities that typhoid

primarily is always an intestinal infection We see in this case that we have another exception to the rule. This case also demonstrates the importance of recognizing the possibility of typhoid meningitis either primarily or secondarily

It is of interest to know whether the infection began in the meninges or in the circulation. We conclude from the history and clinical symptom-complex that it began in the circulation as a septicemia. It is my belief that all cases of typhoid, whether primarily meningeal or intestinal, arise as metastatic lesions from the circulation A fact ever to be borne in mind in the ordinary classical case of typhoid fever is that there is a bacteremia at the time of the appearance of the earliest symptoms, indicating that the blood lesion existed even before or, we might say, in the presymptom stage of the disease

We have seen here the enlarged spleen, mesentery lymph-nodes, and liver Does it occur to you what has occasioned this increase in size of these organs? The pathologist teaches us that it is due to an excessive accumulation of endothelial phagocytes, and not to an ordinary hyperplasia

CLINIC OF DR CHARLES L MINOR

ASHEVILLE, NORTH CAROLINA

ARTIFICIAL PNEUMOTHORAX (FORLANINI) ITS APPLICATION TO THE ARREST OR CURE OF PULMONARY TUBERCULOSIS

Complete Description of Technic. Complications and How to Meet Them. Management of Cases

THE patient I wish to present to you today is a case of bilateral pulmonary tuberculosis whom I have watched for some months hoping that I could rally his resisting powers so that he might be enabled to arrest the disease which has been slowly but steadily advancing in his left lung While some authorities have advocated using artificial pneumothorax in incipient cases of pulmonary tuberculosis I have never felt that this was wise Rational treatment applied in such cases gets such a high percentage of good results that I do not think we are justified in using such a serious procedure as the total putting out of commission of a slightly involved lung, especially when we cannot promise the patient that this lung will finally re-expand and be as good as ever—a thing which occurs in some cases but by no means all It is in obstinately advancing or advanced cases like the one I present to you that I think we have full justification for applying this procedure The process, as I have said is slowly but steadily advancing despite all treatment There is marked evidence of toxemia with all its baneful effects upon the patient's constitution and resisting power and yet the amount of trouble in the better lung is so mild that we can feel reasonably sure that it will safely take up the burden of the double work thrown upon it if collapse is not carried on too rapidly It would seem certain that without some other help this case will go steadily down hill whereas by

using compression, we can offer him, I believe, a fair prospect of arresting this advance and a great probability of stopping the disastrous toxemia which is killing him, unless adhesions prevent complete collapse or some complication causes us to fail Moreover, it may enable us to scar down the trouble in the lung and to return him to working efficiency Statistics have shown that applied to moderately advanced or advanced cases, many of which might reasonably be called utterly hopeless, we can expect from 10 to 20 per cent of satisfactory results, and with care in its application need have no great fear of any mortality from the procedure

The case I present is Mr A C D , age thirty-two, single, occupation bank-clerk, home, New York City His family history is absolutely negative to tuberculosis, "chronic bronchitis," anemia, neurasthenia, or dyspepsia His childhood history from the first to the seventh year is negative From the seventh to the eighteenth year the patient attended day school, and while he had numerous winter colds and tended to be rather thin, he, during this time, had no severe sicknesses and passed as a well boy At eighteen he took up clerical work and has had various bookkeeping positions since, his work places having been usually ill ventilated and dark I might here note that the conditions under which our bank and other clerks habitually work are too generally unpardonably unhygienic Spitting on the floors is not prevented, spittoons are often absent or not used, air and sunlight are at a premium, the hours are long, and in rush seasons overtime is the rule without any extra pay for it That the incidence of tuberculosis under these conditions is heavy is not to be wondered at Some of our larger and more modern banks have most excellent conditions, but the majority of our small banks both in cities and small towns, and of our offices, should be investigated by our Boards of Health

Entering on his present job at twenty-four, he worked from 8 A M until 6 P M , but in rush seasons overtime was often demanded until as late as 11 or 12 P M Three spittoons were all that were at the service of the whole office force and spitting on the floor was necessarily common, several of the clerks having

chronic coughs and expectoration His home conditions were fair and the food good, but he usually had to rush his morning and midday meals His average weight has been 140 pounds, the best in his life 157 pounds, his insurance standard weight should be 165 pounds He denies alcoholic dissipation and lues He admits an occasional moderate use of alcohol Sundays were spent in rest and recreation From eighteen to thirty two, barring the present sickness, he has had no severe illness, but has continued to have winter colds The patient says he was in his usual health until the age of thirty, when his work began to tire him unduly, and he would wake up tired in the mornings and wear out by mid afternoon That winter he had a worse cold than usual and the cough became chronic and was accompanied by a little mucopurulent expectoration, which disappeared that summer Although he was losing weight he paid no attention to his symptoms until the autumn, when one day, without cause, he suddenly brought up about ½ ounce of bright blood The family doctor told him, without physical examination, that this was due to the breaking of a vessel in the back of his throat, so he did nothing for it, but when this occurred again shortly before Christmas he went to the clinic, where a careful examination showed an active process of the upper third of his left lung When I first saw the patient he was a somewhat emaciated man, with unduly bright eyes, a slight flush in the left cheek, a moderate amount of supraclavicular flattening a marked left shoulder droop, and considerable limitation of the motion of the shoulder on breathing The chest was fairly built though a little too long and rather flat. The patient is left handed, yet the tape shows the left side markedly smaller than the right, and the expansion of the left side 1 inch less than the right. The larynx is negative There is considerable pyorrhea, with poor teeth and there is marked obstruction of the right nostril by a spur Percussion shows the isthmus of the left apex to be narrowed, being only 1¼ inches on the left, 2 inches on the right. The motion of the base of the left lung between rest and extreme inspiration is 1 inch, of the right, 2 inches There is dulness L U A to the third rib and a shortened percussion note to the fifth There is

also dulness L U P to the spine of the scapula, impaired resonance to the angle of the scapula In the right supraclavicular and supraspinous fossæ there is slightly impaired resonance Over the upper left lung behind there is bronchial breathing to the second rib, with bronchovesicular breathing to the fourth rib and rather feeble breathing to the base Over the upper left lung in front to the third rib there are medium and fine moist râles, commonly but unfortunately called "subcrepitant râles," and from this point down to the fifth rib patches of crepitations Posteriorly on the left the moisture extends to the spine of the scapula, below which there are fine dry crackles to the angle, with bronchovesicular breathing from apex to midscapula Over the right clavicle there is feeble breathing with one or two very fine dry inspiratory crackles on cough The patient reports a loose, increasingly productive cough on and off all day, with about 1½ ounces of mucopurulent expectoration, chiefly on waking, at which time it is heavier and green The temperature, which is subnormal in the morning, reaches 101+° F by 4 P M, despite rest, falling slowly, until by 10 o'clock it is normal There are no chills, an occasional sweat, the pulse varies from 80 to 110 There is moderate anorexia, with considerable fermentative dyspepsia There is considerable dyspnea on exertion, but none while at rest There is no hoarseness or dysphagia The weight today is 115 pounds While he has made small symptomatic gains during the four months he has been under treatment, the process on the left has extended steadily and the x-ray shadow has shown advance, there being today with the fluoroscope dense shading in the upper left to the third, with cloudlike mottling below extending nearly to the base The right apex is not entirely clear, and there is considerable thickening of the peribronchial glands shadow connected on the right with the apical shadow There are, fortunately, no evidences of pleuritic adhesions in the left lung The motion of the left base, while limited, is not abrogated, and none of the other measures of examination justify a belief in such adhesions As I interpret it, we have here an advancing tuberculous bronchopneumonia in the left lung, with beginning softening of the upper

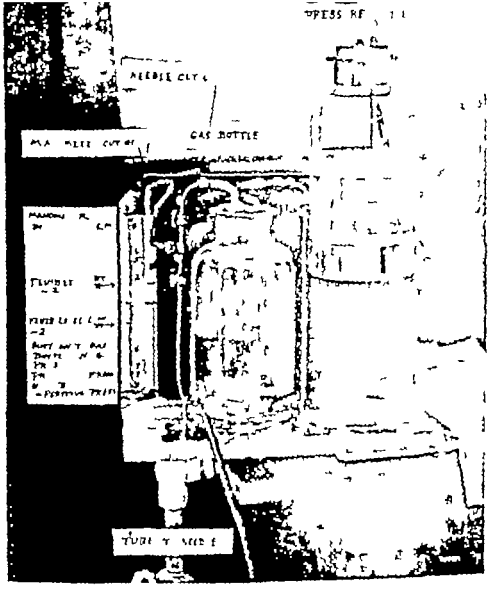

Fig 247

reason to fear any bad results from the use of gas, though should we fail to enter or finally be obliged to give it up as useless the depressing effect upon the patient will be unquestionably very bad If, as is reasonable to expect we collapse his left five or six fillings, and keep it collapsed thereafter for the next six months to two years it is not

hope that he may be restored to working efficiency within the year, as I have seen happen in a number of cases Should he not be able to remain under my care the refills can be done at his home by any intelligent physician who will supply himself with the necessary apparatus and follow a strict routine in his procedure Having then decided to carry out the pneumothorax, I shall proceed to prepare my apparatus I will place this table near the patient's bed, cover it with sterile towels, and

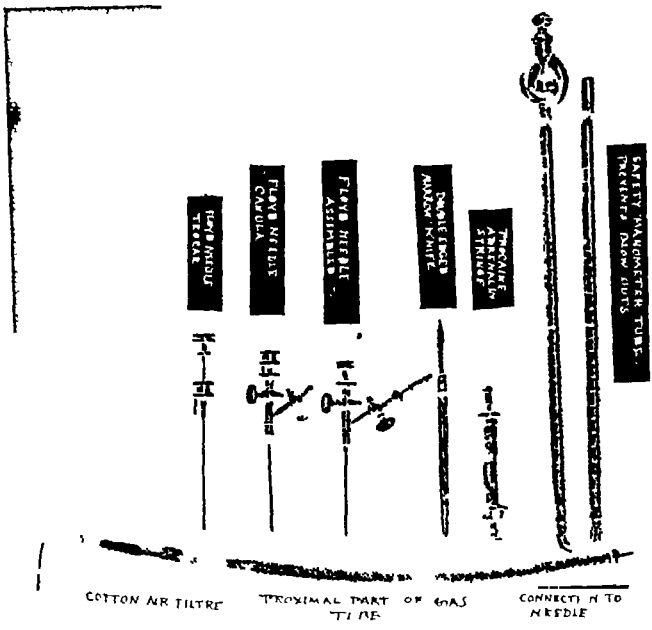

Fig 248

place here two basins of solution, one for my hands and one for the cotton sponges, of which we will only need a few On the corner of the table near the patient, so that if I have no assistance I can reach the gas cocks easily, I will place the gas bottles, so faced that the manometer will be in full view as I work, as a knowledge of its fluctuations is *essential* to safe and sane procedure An assistant is a great convenience, but is by no means indispensable On the sterile towels I lay out the needle, that

of Robinson being the one that I have always used and which I have found entirely satisfactory This needle and the apparatus are made by Codman & Shurtleff, of Boston, by Max Wocher, of Cincinnati, and by other instrument dealers While there are numerous other arrangements of gas and pressure bottles for the purpose, and the Floyd & Robinson apparatus is only one of many, I have found it extremely handy, easy to carry from house to house, simple to manipulate, and the arrangement of cocks for throwing in or out of connection the manometer or gas tubes is extremely convenient. The arrangement also for raising or lowering the pressure bottle is satisfactory My only criticism is that the manometer is rather too short, so that if the patient coughs while it is open he will at times blow out the water A very satisfactory apparatus can be improvised at small cost by any doctor with two 2 liter graduated glass bottles, perforated corks, glass tubing, cut-offs, rubber tubing, and a manometer, but it is usually more convenient to buy the apparatus ready made. I formerly always used nitrogen gas, but various workers, notably Webb, have shown that air is just as satisfactory, and that anyway, after a short time, the gas in the pleural cavity shows the composition of air, so that now, having run out of nitrogen, I have used air with entire satisfaction I have filled the gas bottle with air by first filling it full of bichlorid solution and then lowering the pressure bottle to start siphonage, letting the air pass through cotton to filter out any dust. Along with the needle I lay out on the towel this small narrow double-edged knife, also this Luer syringe containing 1 or 2 per cent. procain (formerly called "novocain") solution in 1 10,000 adrenalin and have handy another syringe with morphin and strychnin tablets for use in case of necessity The gas tube and glass-cotton air filter should be dry sterilized while the water in the bottles should contain 1 1000 bichlorid or cyanid of mercury

In choosing the point to enter we naturally wish to go in where there are as few adhesions as possible, and this demands of course, a very full knowledge of the percussion and auscultation findings of the lung as well as an x ray plate or a fluoroscopic

examination There are no very satisfactory means of determining where we will meet adhesions Sometimes stereoscopic plates are useful and sometimes auscultation will help us, especially where abundant frictions reveal old pleurisy, but I have found percussion my best guide, and have rarely failed to get in where I have gotten a good clear percussion note Further, it is essential that we be thoroughly informed of the condition of the better lung, so as to be sure it can bear the burden of the double work it will have to take up, and so as to be familiar with its former condition and able to compare it with its condition after the pneumothorax has thrown an extra tax upon it Where feasible, I find it distinctly best to avoid the popular point of entry in thoracentesis, namely, the ninth interspace below the angle of the scapula, and to enter the needle somewhere between the nipple line and the midaxillary line, usually in the fifth interspace When going in on the left side, as in this case, it is very important to be sure that the heart has not been dislocated to the left, as it is by no means impossible, through carelessness, to puncture it, and hence it is a wise practice always to mark the apex-beat before proceeding with pneumothorax In the case in hand I find normal resonance all over the area between the nipple and the midaxilla, and that the base of the lung has 1 inch of good motion on deep inspiration and the heart apex is in its normal place In this area I find today a few crackles (pleural?) on cough, as well as a little cog-wheel breathing, which may indicate a slight pleurisy, but probably not enough to hinder the operation I will now place the patient on his right side with a pillow under it so arranged as to make, if possible, the the chosen point of entry the highest point of the lung, so that our bubble of air will remain there and not tend to travel to some higher point However, while this is desirable and correct, I have often gotten in easily where it was impossible to raise the point of entry to the highest level I will next prepare the field which may be rendered aseptic by any of the recognized methods Personally, I have found green soap, alcohol, and mercuric cyanid solution entirely satisfactory, as it leaves the skin moist and pliable, whereas iodin makes it abnormally dry and harsh I will

now surround the field with sterile towels and will raise the patient's left hand well over his head both to assist in separating the rib spaces and to leave me a clear field (Fig 249) Now with sterile fingers, I feel for the chosen interspace and with a hypodermic full of the anesthetic infiltrate a small area intradermically then plunge the needle in vertically to the pleura, on which I deposit a portion of the syringe full of the anesthetic, depositing it as I withdraw my needle, in the intervening tissues In very nervous patients it may be necessary to use another

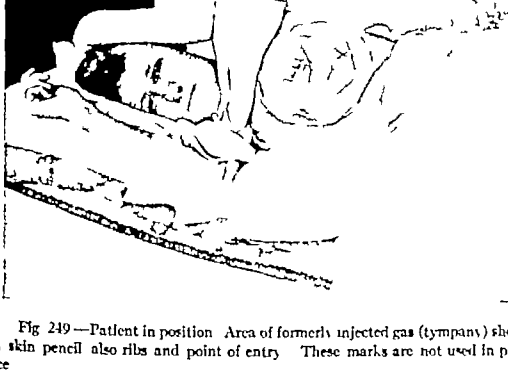

Fig 249—Patient in position Area of formerly injected gas (tympany) shown by skin pencil also ribs and point of entry These marks are not used in practice

ful and in any case we should let enough time elapse so that the anesthesia is complete and there will be as little pain as possible, as pain makes the patient nervous and makes the operation more difficult Indeed where the pain is severe and the patient tends to hold his breath and press down, he may so inflate the lung against the chest wall as to make it impossible to get air into the cavity When done carefully the procedure is usually entirely painless, though sometimes nervous women complain of suffering a great deal which suffering[1] I am satisfied is much

[1] Note facies of patient in Figs 251 and 252 showing no pain or anxiety

more in their minds than in their bodies I will now take the double-edged knife and make a small epidermal incision, which greatly facilitates the entry of the rather obtuse needle In cold weather it is wise to place a part of the gas tube in a pitcher of hot water to warm the air or gas Now taking the needle, holding it as I show you with the top in the palm of my hand and its shank between my thumb, index, and middle fingers, so that its axis of entry will be in the axis of motion of my forearm and its point entirely under control, so that its advance can be stopped

Fig 250 —Patient and instruments ready Note method of holding needle, butt in palm of hand, shaft held by thumb, index and middle fingers, giving proper control of advancing point

quickly, I pass it through the small skin opening, through the intervening tissues, going through the interspace down to the pleura, and if you listen I think you will hear it pop through the fascia or pleura, which of these tissues it is that makes the sound I cannot say, pleura or fascia, both have been claimed by different writers To me it seems as though it must be a combination of the two In any case it is usually immediately after we pass this point that we manage to get into the pleural cavity Before going further let me advise you not to hold your needle, as some do, like a pen, depending upon wrist motion for its entry It is

much more difficult in this way to stop the advance of the point and you are, therefore, much more apt to wound the lung (Figs 250 and 251)

As you will observe, I have entered the needle while it was shut off from the manometer, because at times a high negative pressure may draw the fluid out of the tube chestward My needle point should now be so located that when I open my manometer I will promptly get negative fluctuations of at least

Fig 251 —Needle has just been entered manometer reads —2 —6 Gas has been turned on and is flowing easily hence pressure bottle is not elevated. Needle is steadied at point of entry by index and middle fingers of left hand protected by wet cyanid cotton Shank held by right hand Trocar withdrawn and shut off from cannula by cock It can be reintroduced if needle becomes obstructed

2 or 3 cm , smaller fluctuations than this, from $\frac{1}{2}$ to 1 cm are not to be relied upon as a guide You will observe that having opened the manometer I get no fluctuations at all This either means that my needle has not passed the pleura which I am certain is not the case, since I heard it "pop," or that it is engaged in thick adhesions I will now push the needle very slowly forward, keeping the manometer open I note that the needle point does not feel free, and it will be impossible to be sure that I may not puncture the lung You may notice that I am getting

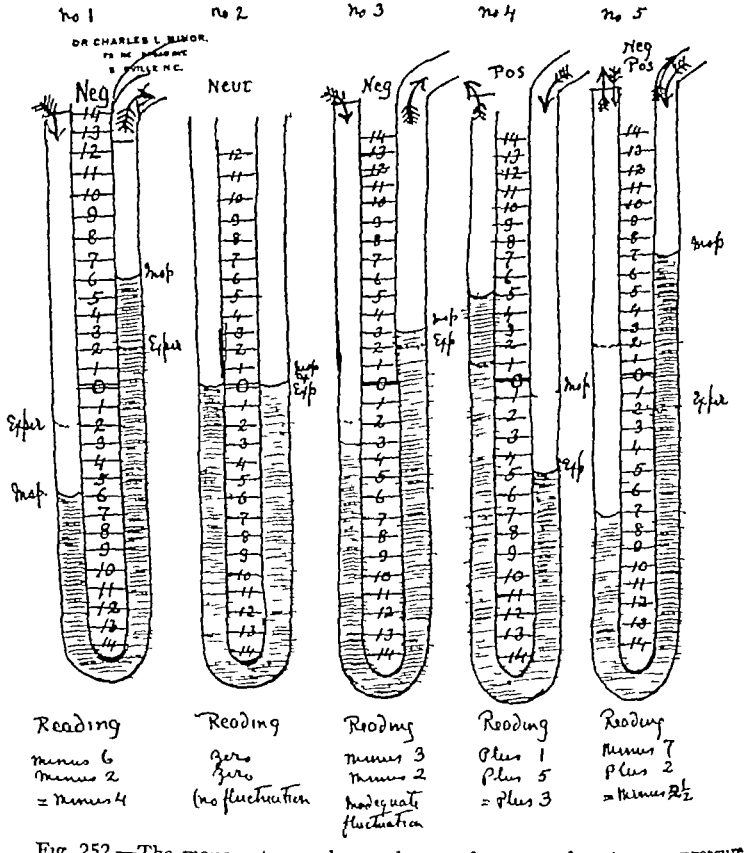

Fig 252 —The manometer reading indicates the state of suction or pressure within the pleural cavity One end of the manometer being connected by tubing and needle with this cavity, as long as breathing goes on we have a regular rise and fall of fluid in the two legs of the manometer When there is a vacuum in the chest we get suction which raises the fluid in the thoracic leg of the manometer and naturally depresses it in the other leg We read the level of motion at inspiration and expiration, the correct reading being the mean of these two readings Where by introducing gas into the thoracic cavity we get pressure, we then begin to get positive readings, the fluid being depressed in the thoracic side of the manometer to below the zero point and raised on the other side Usually in a first injection we get free negative fluctuations running between —9 and —2, showing the existence of a strong vacuum in the thoracic cavity As the gas flows we get less and less vacuum and suction, until toward the end of the injection we may get a reading, for instance, such as —7 and —4 If reinjections are postponed too long we will find that we get marked negative readings at these reinjections, whereas, if they are spaced wisely, we will still find slight positive or mild negative readings. The

moderate, positive, equal fluctuations and know, therefore, that my needle has gotten into lung tissue. This is unquestionably undesirable, as you may thereby infect the pleural cavity, yet it occurs from time to time and usually has no bad results, though sometimes the patients will spit a little blood immediately thereafter I have now tried several intermediate points, advancing and withdrawing the needle carefully, and have failed entirely to get proper free negative fluctuations, I will, therefore, remove the needle and seek a better point of entry I find that more than two trials at entry is apt to make the patient extremely nervous, hence if you fail twice in succession it is best to postpone another puncture to a later date. This point, 1 inch outside the nipple, gives good resonance, the breath sounds here are clear, and, as I have already shown you, the motion of the base, while limited, is not absent, and the apex beat is not too near Having prepared the new field, I will take a new sterilized needle, lest the first should have become infected, and prepare for another entry You will note that the patient winced a great deal at my incision, which proves, I am afraid, that I was rather too impatient and did not give time enough for the procain to take full effect. There is a certain danger in this, as it makes pleural shock much more possible, indeed, if the pleura is always carefully anesthetized, such shock would seldom or never occur I think that you have again clearly heard the point pass the pleura. I open the manometer, and you now see that I get satisfactory negative fluctuations from -4 to $-6\frac{1}{2}$ cm , and I now feel perfectly safe in opening the gas-cock and shutting off the manometer Some consider it important to keep the manometer connected with the pleural cavity while the gas is flowing, but if you take readings after each 100 or 200 c.c. of gas has entered, I believe this to be unnecessary, and if left open a sudden

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fluctuations, to be a satisfactory guide, must be greater than 1 cm. In early injections and possibly in any injections it is unwise to produce too great a positive pressure, and we must be content to go cautiously and not to change the pressure too greatly at one time. Incidentally I might state that the fluid in the manometer should be colored with some dye so that its level can be quickly and easily read

cough will empty your manometer At times we get a single high negative rise which remains stationary or falls slowly to zero Saugman ascribes this to the closure of the point of the needle by contact with the visceral pleura Watching the level of the fluid in the pressure bottle and observing its relation to the graduations on the bottle, which are in centimeters, you note that the water steadily falls as it presses the gas in the gas bottle into the pleural cavity, 100 c c now having entered, I will shut off the gas and measure the pressure You will observe that the pressure has risen considerably with so small an amount as 100 c c of gas, the reading now being -3, -5 This relatively large rise with a small amount of gas would indicate that the space into which the gas is flowing is rather restricted, which may be due to unbreakable adhesions or to slight delicate ones, in which latter case, if I continue to inject gas with some rise of pressure, they will probably break down and the pressure will become more negative We have now put in 200 more c.c, which makes 300 in all, which is as much as I ever allow myself to put in at a first injection, though it would be easy to inject a much larger quantity You will note that the patient complained of a slight pain during the injection, and I think you will find that we have broken some slight adhesions Slight pain during the procedure can be neglected, but severe pain is an indication to stop for that time Reading the manometer, you note that despite having put in twice as much as at the first flow, our reading is $-2\frac{1}{2}$, $-4\frac{1}{2}$, and I doubt not the gas has burrowed its way through slight adhesions and formed a very decent bubble, else we would have gotten a more marked rise There is a great temptation to go ahead and put in more, but experience has taught me that it is much wiser for the first time to be content with a small quantity, hence I will now draw out the needle. When I first applied the procedure I followed Brauer's advice and put in as much as 1000 to 1800 c.c of gas, but as a result caused at times waking up of sleeping processes in the good lung Since then I have followed Forlanini's rule of not over 300 c.c, and have had no such trouble Now with a pledget of moist sterile cotton I rub the puncture hole in various directions so as

to assist in the closing of the channel We will now place a small square of sterile gauze over the puncture and, since the final pressure was not high, will put only a simple adhesive over this If it were a reinjection and I had used high pressures, with the probability of gas leaking out through the canal and consequent subcutaneous emphysema, I would put a small firm ball of cotton over the gauze at the site of the puncture and strap down firmly on this This is usually sufficient to prevent leakage. You will note that the patient has had little or no pain and is in perfectly good condition I find his pulse excellent, and it will not therefore be necessary to give him a hypodermic of morphin In nervous patients when I have not, as is my habit with them, used it at the beginning of the procedure, I sometimes give it at the end to quiet the patient down for the next few hours and prevent cough, which favors emphysema After a first injection I prefer to keep the patient quiet in bed all day

In refills it is usually perfectly safe for the patient to get up at once and go out again on the cure, if he be afebrile, and on exercise, which is rarely the case at first with the patients to whom I apply it, but some authors advocate bed rest in all cases You will have noted that the gas and pressure bottles in this case I kept upon the same level. This was because it was a first injection and I did not wish to use any very high pressure but usually it is desirable to raise the pressure bottle so that the pressure of the fluid will make the entry of the gas easier This is especially necessary where there are many adhesions which we may desire to loosen or where the gas begins to flow in very slowly showing too much resistance in front, but it must not be forgotten that in the use of very high pressure there is a possible danger of rupture of the mediastinum, with the production of a double pneumothorax. Another great danger of high pressure is the tearing of an adhesion from its lung insertion, thus infecting the pleura and forming a pyopneumothroax. If you will observe the patient you will see that he shows no dyspnea or discomfort of any sort (See Fig 251) Dyspnea is rarely felt after the first injection, but when we use larger quantities later on it is sometimes slightly troublesome and may demand the removal of part of the gas but

I have never seen any serious results Gas can be removed by starting siphonage from the pressure bottle, and as the antiseptic solution flows out it sucks out a corresponding amount of gas A sense of tightness in the chest, however, is usually produced at each injection of gas, and, indeed, the patients are not satisfied after they have had a number of injections until they feel this tight feeling They seem to feel uncomfortable when they feel "loose," as they call it, and this sense of looseness, with return of cough and expectoration, are our best indications for a refill Of course, we should not wait for the development of such symptoms, but should so space our refill as to anticipate their appearance At the first few injections we rarely reach a positive pressure if we have been prudent and gone forward cautiously At later fillings, however, it is desirable to bring about positive pressures There are some workers in this line who aim never to pass zero I myself prefer moderate positive pressure, but do not like to go higher than 4 cm of water, though some very good authorities (Riviere) have used much higher pressure than this On the whole, I prefer in refills that my final pressure run between zero and 4 cm If pressure is not high enough, refilling will become necessary too soon, but if it is too high there are, as I have already stated, certain dangers connected with it, the greatest of which probably is throwing the burden of extra work too suddenly on the other lung, which, you must remember, usually has some foci of trouble in it, and if it is too quickly forced to compensatory action these may very easily light up into activity High pressure also may, as I have said, rupture the mediastinum or produce troublesome deep or subcutaneous emphysema, which is not a serious complication but a very uncomfortable one, and which also prevents any useful subsequent auscultation You will note that during the procedure I held the needle at the point of entry steadily by the fingers of my left hand (see Fig 251), interposing, however, a pledget of wet cyanid cotton between the skin and the needle to decrease the possibility of any infection, and was careful to hold the shaft of the needle by my right hand as immovable as possible, since an excessively small advance or lateral motion will often run the point against some adhesion and stop

the flow, or, what is much more dangerous, may force the point through a distended vein in an adhesion and produce that chief and worst complication of this procedure—air embolism. A wabbling needle point, therefore, is a distinct danger and must be guarded against by a very steady hand, and for this reason it is much better to have an assistant, so you can put all your attention in keeping the needle point still I would again stress the extreme importance of proper holding of the needle on entry, the butt being in the palm of the hand This allows great delicacy in advancing and withdrawing the point and gives a certainty of control obtainable in no other way Should you at any time have the misfortune to enter a blood vessel, you will find the blood showing itself in the small glass cotton filter tube which you see I have placed in the course of the rubber gas tube, and you will note in the manometer a rapid rise of pressure with out fluctuations If by any possibility you had not observed this and had opened your gas-cock you would very probably have killed your patient If, on the contrary, the gas is not turned on and the needle is quickly withdrawn no harm will occur While it is a safe and wise rule never to open the gas-cock until you get distinct negative fluctuations, I have in certain cases violated this slightly when I have been sure my needle was in the right place, and yet when I could get no fluctuations to speak of, by quickly opening and shutting the gas-cock for the fraction of a second This admits a minute bubble of air and often forces apart the pleural layers sufficiently to give you at once clear fluctuations, and I cannot believe that the very minute amount of gas that is thus introduced can possibly do harm

In the case I have shown you I expect to follow my usual procedure and renew gas on the day after tomorrow, entering not through exactly the same puncture hole, but in the immediate neighborhood My third injection I usually give on the fourth day, my fourth after a week, my fifth after ten days, my sixth after two weeks, my seventh after three weeks, my eighth after a month, but, of course, cases vary and no hard and fast schedule can be followed in all cases Sometimes I allow two months to elapse, but usually a month is the longest interval that is safe

However, I am becoming more and more convinced, with Riviere, that more frequent refills are better and prevent re adhesions, and believe two weeks is often wiser than four

The question of readhesions is one of the troubles of the procedure, and brings not a few cases to an unsuccessful termination after weeks or months of great success How it occurs I am not prepared to say One would think that if the collapse had been satisfactory it would be impossible for the parietal pleura to touch and adhere to the visceral, but the fact remains that sometimes after introducing gas with no trouble at all the space begins to get smaller and smaller, it is more and more difficult to get in any gas, and finally the attempt has to be given up as a failure This may usually be avoided by shorter periods between refills, giving the pleural surfaces no chance to touch and readhere I have one such case at present that is going down hill, with waking up of the process in the partially collapsed lung where, had I been able to continue to introduce gas, I feel sure that he would have made a complete and satisfactory recovery For this reason it is not safe to stop the gas for two or three months and then try to recommence While in some cases this may be perfectly possible, it is frequently a failure for the reasons stated.

When once the lung has been well collapsed, it is often perfectly satisfactory for the patient to take up regular work while continuing to receive injections, and it is reported that certain pneumothorax cases are fighting in the German Army

You may ask what course I would expect this case to follow if it behaves normally? In a perfectly and properly collapsed lung you will find the temperature drops rapidly, although in some nervous and excitable people there is a slight rise at first, followed by a fall later The cough and expectoration are temporarily increased, while the patient spits out the sputum which is forced out of the collapsing lung, but this is followed by a remarkable and most satisfactory decrease, and very soon all expectoration ceases The psychic effect upon the patient of his wonderful improvement is splendid He feels hopeful and, getting rid of the terrible toxemia in one or two weeks, he feels like a new man. The tiredness which oppressed him disappears, the light of health

comes back into his eyes, he is freed from most of his trying symptoms, and has to be carefully watched lest he become too enthusiastic and foolishly overdo In a few left side cases the diaphragm is pressed down upon the stomach the appetite is affected and dyspepsia produced, but this has not been frequent though once or twice a troublesome complication The promptness of the improvement in many cases is one of the wonders of

Fig 253—Another case of good compression of left side Basal and apical adhesions prevent complete collapse but center part is well compressed toward mediastinum Heart shoved over to right side

therapeutics and can be compared to nothing so well as the effects of the successful use of salvarsan To see a very sick man suddenly changed to one who feels well to see the racking cough stop, the profuse expectoration after a temporary increase disappear, to see the fever fall the weariness leave him the eyes brighten—is a very delightful experience to any doctor In the best cases this improvement continues uninterruptedly until the patient is able to return to his work In others it is inter

rupted by certain complications, the most troublesome of which is the collection of fluid This I have come to look upon as an unfavorable occurrence, although at times fluid takes the place of gas with perfect satisfaction and remains uncontaminated but in a certain percentage contamination occurs, and when this occurs we have a dangerous complication on our hands

I have already referred to the readhesion of the lung, a most disappointing complication One of the saddest things connected with the procedure is the very painful effect upon the patient, who has begun to see health coming back by leaps and bounds, when, through some complication, the procedure has finally to be given up It is hard, indeed, for the doctor then to make the patient bear it patiently or calmly, and the absolute loss of hope hastens the fatal end

I failed to note that it is very wise at the end of the operation to map out carefully with the skin pencil the area of tympany which marks out the gas bubble and also to note the displacement of the apex of the heart and of the mediastinum, and to examine the patient with the fluoroscope At successive refills it is very interesting to watch the fingers of gas push themselves out in different directions between the layers of slightly adherent pleura One would expect the introduction of the gas would at once prevent the transmission to the ear of any adventitious sounds from the lungs, but it is not always the case, and it is often surprising how large an amount of compression can be brought about before we entirely obliterate such sounds The reinjections, since there is already existing a good gas pocket, are usually extremely easy, but it is well to remember that air embolism has more frequently been reported at reinjections than at first injections, probably on account of the needle entering a vessel in some adhesion, and therefore we must continue to be as extremely careful as at the first introduction

This patient must now be kept quietly in bed for twenty-four hours at least even had he been afebrile Since he was a febrile case he will be kept there in perfect quiet until the procedure has largely freed him from temperature, something which I hope we may anticipate within a week or so

Subsequent Note (two months later)—The collapse has gone ahead very satisfactorily as far as the lower two-thirds of his lung are concerned The fluoroscope shows it well compressed against the mediastinum from the base up to the level of the third rib From this point up to the apex the adhesions have prevented a complete collapse, and it is unlikely it will be possible to break down such dense adhesions, but the patient has benefited greatly, his temperature has ceased, his cough and expectoration, while not all gone, are very much lessened, the toxemia has disappeared, and the general condition is much better He has gained 15 pounds in weight, though many of these cases lose rather than gain after the operation, probably owing to the lessened area of oxidation It is very seldom that the lung undergoes a perfect collapse. In one case which I had it was collapsed to the size of a tennis ball, and the shadow protruded from the right side of the sternum between the third and the fifth ribs, the whole of the rest of the pleural cavity being clear In many other cases bands of adhesions show up very plainly, running out in star like radiations to the walls and giving butterfly like shadows Judging from his present condition this case is going to get a satisfactory result, but, of course, it is impossible for me to say how long I will wish to keep up the reinjections of gas Many patients never wish to stop, their relief being so great and the terror of relapse so acute that they prefer the monthly reinjections to the chances of a normal expansion, and I can scarcely blame them, for I have seen renewal of activity after such expansion, and usually this means a fatal result. So far we have not data enough to guide us in forming an opinion on this question but, of course, the ideal will remain a complete re-expansion and resumption of normal function, but many men, from fear of a relapse or at their patient's request, continue refills indefinitely In this connection the remark of an intelligent medical student who had seen a case which had been under pneumothorax five years seems to me to cover the ground "It seems to me pathologically unsound to do it, though perhaps it may be clinically correct."

CONTRIBUTION BY JAMES S McLESTER, MAJOR M R. C

BIRMINGHAM

CHIEF OF THE MEDICAL SERVICE, BASE HOSPITAL, CAMP SHERIDAN, ALA.

CERTAIN MEASURES TO PREVENT THE DISSEMINATION OF DISEASE

WHILE much has been written recently concerning the modifying influence of war upon modern surgery, it will probably be found that we shall also have to revise in a radical manner our works on medicine The chapter on pneumonia must be entirely rewritten, indeed, we can truthfully say that it has been largely rewritten within the past three months

It is impossible to discuss here all of the new things which we have learned since the intimate contact of hundreds of thousands in the army camp has offered such fertile fields for the spread of disease. The rapid spread of respiratory infections, the clinical types seen, the high mortality in the post measles bronchopneumonias, the part played by the Streptococcus hemolyticus, the frequency and the high mortality of empyema with its surprises at necropsy, all are of great interest, but we have had as yet insufficient time for their satisfactory consideration.

From all our rich experiences of the past winter, however, there is one thing which we seem to have learned fully and finally, that is, the value of preventive measures To me this has been our greatest lesson, and it is of special value in that it is applicable to the civil hospital as well as to the armed camp If we are to prevent the spread of respiratory and other contagious diseases three measures are necessary (1) the avoidance of overcrowding, (2) the universal use of the cubicle method of isolation, (3) the employment of masks for patients and attendants

Pneumonia is the one disease that we fear most and toward which preventive measures are chiefly directed, but the same means which will prevent the spread of this disease are applicable also to scarlet fever, meningitis, diphtheria, measles, influenza, tonsillitis, and many other diseases In fact, when we consider the "droplet" as a means of disseminating these diseases, we come to wonder how great a proportion of all bacterial invasions occur in this manner, and are tempted to extend widely the application of our preventive measures

The necessity for ample floor space between beds and for plenty of air is obvious In the Base Hospital at Camp Sheridan we have escaped the great pneumonia epidemics which some of the camps have experienced, and all types of pneumonia seen by us have been mild, with a relatively small incidence of empyema. We have attemped to explain this fortunate circumstance in various ways, but after comparing our experience with that of certain other hospitals I am convinced that the ample floor space which we have been able to allot each patient, and our consequent escape from overcrowding, have been the most important factors We have endeavored always, particularly in pneumonia, measles, and scarlet fever, to provide 1000 cubic feet of ward space for each patient

The soldiers with acute lobar pneumonia have been placed on the veranda, with universally good results, and many of the patients with bronchopneumonia following measles or influenza have also been treated in the open, but there seems to us to be a difference in the way in which these two groups of patients take the "open-air" treatment. During cold, disagreeable weather the patient who has developed bronchopneumonia as a sequel to bronchitis or measles certainly seems very miserable and unhappy on the veranda It is difficult to make him comfortable and his cough is at times very annoying The explanation of this difference may be sought in the fact that the one is suffering with a disease of the lung, pure and simple, and the other, in addition, has an irritable upper respiratory tract We have become convinced that the open-air treatment can sometimes be carried too far, and that for certain patients ample space in a well-ventilated ward during the winter is frequently to be preferred

The space allowed each patient should never be less than 1000 cubic feet, and continuous free ventilation of the entire ward should be assured This is a measure of infinite importance.

The surgeon general directed early in the winter that the cubicle system of isolation be adopted in those wards in which patients with contagious diseases were being treated Wires were stretched across the entire width of the ward, 6½ feet from the floor, between the beds, and sheets were hung upon these wires in such a manner as to isolate each bed in a sort of cubicle. The space below and above the sheet permits the free movement of air, but no patient can by talking, coughing, or sneezing throw his bacteria laden droplets of moisture to the bed of his neighbor The evident effect of this barrier in preventing the transference of disease from one patient to another offers eloquent testimony as to the great rôle played by the droplet in spreading contagious diseases

In certain instances the influence of the cubicle was remarkable At a time when we had a ward full of patients suffering with measles, and everybody was coughing and sneezing respiratory infection was almost universal among these patients. True, as soon as a case of bronchopneumonia was recognized the patient was transferred to another ward but in spite of this precaution the dreaded complication increased Finally, more room was provided for the individual patient and each bed space was converted into a cubicle The coughing and sneezing immediately ceased and the bronchopneumonia disappeared The transformation was graphic. This marked the end of bronchitis and pneumonia as a dominating factor in the measles ward and the lesson is not easily forgotten

In April we were overtaken by an epidemic of so-called influenza, and the number of patients in the hospital suddenly increased from 400 to 900 It became necessary to place these soldiers in wards with patients suffering with other affections and these latter began rapidly to come down with influenza. Because of this we then decided to extend the cubicle system to all the medical wards without exception and the beneficial effect of this measure was immediately felt. As illustrative of its influence, take our experience in a certain ward Not until late

in the epidemic was it necesssary to place any of the grip patients in the psychopathic ward, at which time we had already instituted the general use of the cubicle, and as a result, contrary to our earlier experience in other wards, none of these psychopathic patients developed influenza

The hospital of the future, both civil and military, must make provision for the protection of its patients by placing every bed in a cubicle It may be that fixed partitions or movable screens will be found preferable to sheets hung upon wires, but some such system will come Another winter in the base hospital will, I believe, find all the medical beds in cubicles

One more means of protection has been found of real value. The wearing of masks by physician, nurse, attendant, and pa tient It has been our custom to require masks of physicians, nurses, and attendants in all contagious wards, but the experience of Capps at Camp Grant has caused us to extend this measure to all patients under quarantine who are out of bed A certain "miscellaneous" ward, for instance, is quarantined because of the appearance among the patients of a case of scarlet fever or diphtheria Everybody knows how frequently the period of this quarantine must be repeatedly extended because of the appearance at intervals of still other cases, but such "cross-infection" can be entirely eliminated if Capps' suggestions are followed, and each patient wears a mask when out of bed except when alone in the toilet or bath I am sure that by means of the mask we have limited the complications in our contagious wards

The mask consists of two layers of gauze cut square, with a piece of tape sewed at each corner, and is sufficiently large to protect the nose and mouth In this recent epidemic I wore a mask constantly when making the rounds of all wards, and it is the first time I ever passed unscathed through a grip epidemic. Other officers had a similar experience For my own protection I would not today think of examining a pneumonia or a meningitis patient without a mask, and if none were at hand would protect my nose and mouth with a handkerchief

The value of these preventive measures has been forcibly borne in upon me, and I write of them here because of their importance to the hospital of the city as well as of the armed camp

CLINIC OF DR BRYCE W FONTAINE

Memphis General Hospital

A CASE OF ACHONDROPLASIA

History—White man Age thirty four Telegraph [illegible] by occupation Height, 51 inches Weight, 100 pound [illegible] mitted to hospital because of nausea and vomiting which he has existed for five days Temperature, 98 4° F Pulse 90

Physical Examination.—Thoracic viscera negative Abdomen shows slight tenderness over epigastric region Blood Leukocytes, 8000 Differential count normal Urine negative The condition for which this patient enters the hospital is an acute gastritis which has resulted from imprudent eating and is uninteresting to us compared with the man's physical development Mr A, examine this man's body and limbs, and tell us what you think of his physical development

Mr. A I find that the man is a dwarf that he has a normal head, has an expression of intelligence, has a trunk of normal proportions has normal sexual organs, but has very short arms and legs

Dr. Fontaine What would you call such a condition as this?

Mr. D Cretinism

Dr. Fontaine That is exactly the answer I hoped you would give, because these cases are nearly always mistaken for cretinism Mr D in what way do you think this man resembles a cretin?

Mr. D In his size

Dr. Fontaine In this I think you are mistaken because most achondroplastics are taller than the average well-developed cretin An achondroplastic may, however, resemble a cretin but the mistake arises from the mere fact that he is a dwarf

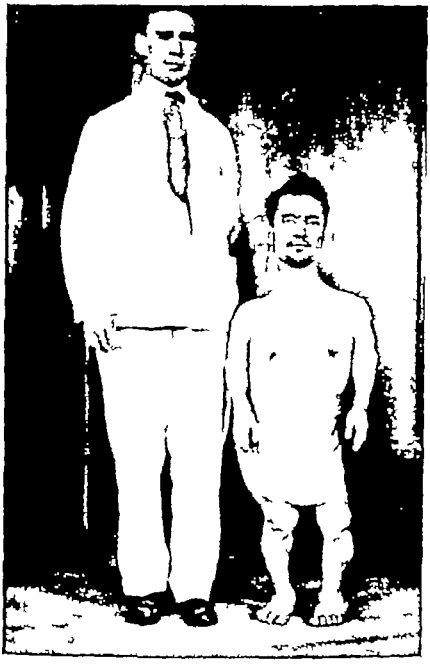

Fig 254 —Achondroplastic compared with man of average size.

Fig 255 —Trident hand

DR. FONTAINE Yes, two other diseases, rickets and osteogenesis imperfecta Rickets in its ultimate results usually de

Fig 257 —Roentgenogram of forearm, showing abnormal curving of radius, with enlarged ends of radius and ulna

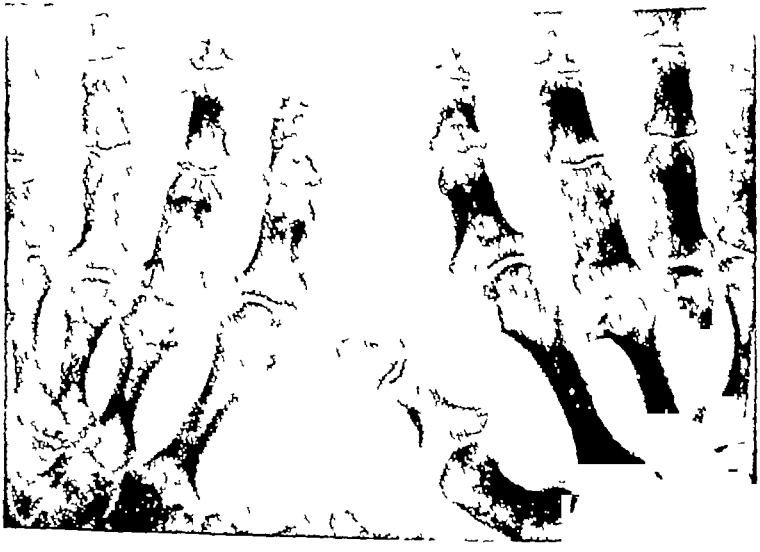

Fig 258 —Roentgenograms of hands

forms and dwarfs the victim until, at first sight he looks very much like an achondroplastic The distinguishing features are,

however, that all the bones of the body are affected in rickets the ribs, head, spine, and pelvis The long bones are usually much more deformed, bent, and twisted out of shape than those of an achondroplastic In cases where the diagnosis is difficult we can always distinguish the two conditions by means of roentgenograms

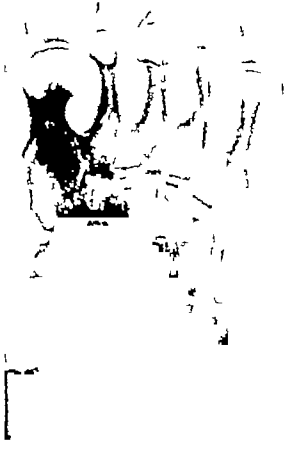

Fig 259—Roentgenogram of foot

Osteogenesis imperfecta occurs so rarely that it need not give us much concern in the differentiation This peculiar disease results from imperfect ossification of the long bones and as a result, repeated fractures of the limbs occur causing dwarfness and deformity It is very rare for cases of osteogenesis to survive

until adult life, and in the few cases that do, the roentgenogram will clearly show the line of fracture and union in the long bones thus differentiating it from other conditions

Here we have a dwarf with the peculiar development of achrondroplasia The photograph of the patient shows his size as compared with a man of average height A striking characteristic of the disease, in addition to the short limbs, is the presence of short chubby fingers, all of the same length, and the abnormal separation of the ring and middle fingers, constituting what has been termed the "trident hand" (see Fig 255)

Examination of the roentgenograms of the long bones shows the diaphysis and epiphysis broadened and more plump than normal, and the middle of the long bones correspondingly narrower The normal curves are all exaggerated Roentgenograms of the hands and feet show the same broadening at the ends of the small bones

The etiology of this disease is as curious as it is interesting after it has developed It has been known to have presented a family characteristic Males are more frequently affected, and it occurs as a result of some developmental disturbance during fetal life, causing a premature closure of the epiphyses

The disease bears no relation to syphilis, rickets, or any disturbance of the ductless glands, and is absolutely unaffected by any medical treatment

CASE I.—Patient W S Male. Negro. Age twenty four, 5 feet 6 inches tall. Weight, 130 pounds Occupation, laborer

Chief Complaint.—Distention and pain in abdomen, some nausea, vomiting, and dyspnea

Present Illness.—Began six weeks ago, with pain in abdomen, lasting a few minutes at a time, but continuing both day and night. Within the last week patient has lost flesh and strength, has had fever and anorexia.

Previous History.—Negative. Habits good Ordinary diseases of childhood Denies venereal diseases.

Family History.—Negative for tuberculosis.

Physical Examination.—Body well developed, poorly nourished

Eyes.—Sclera and conjunctiva bile-tinged Mucous membranes anemic.

Throat.—Negative.

Tongue.—Thick frosty coat over dorsum.

Teeth.—Caries and pyorrhea.

Heart.—Apex beat in fifth interspace, 9½ cm from median line. Heart sounds clear throughout. Pulse regular

Lungs.—Normal vesicular breathing throughout. Percussion note resonant throughout A few small crackling râles can be heard over base of right lung posteriorly

Abdomen shows distention of moderate degree, circumference at umbilicus 100 cm Percussion shows tympany in upper part about umbilicus, dulness in flanks. Fluctuant wave distinct Patient in sitting posture shows flatness in lower part of abdomen as high as umbilicus, tympany above Palpation of abdomen elicits distinct tenderness, and in several areas there can be felt masses of varying size and shape. Because of the ascites it was impossible to palpate the liver or spleen

Muscles.—Soft and flabby Lymph nodes not palpable

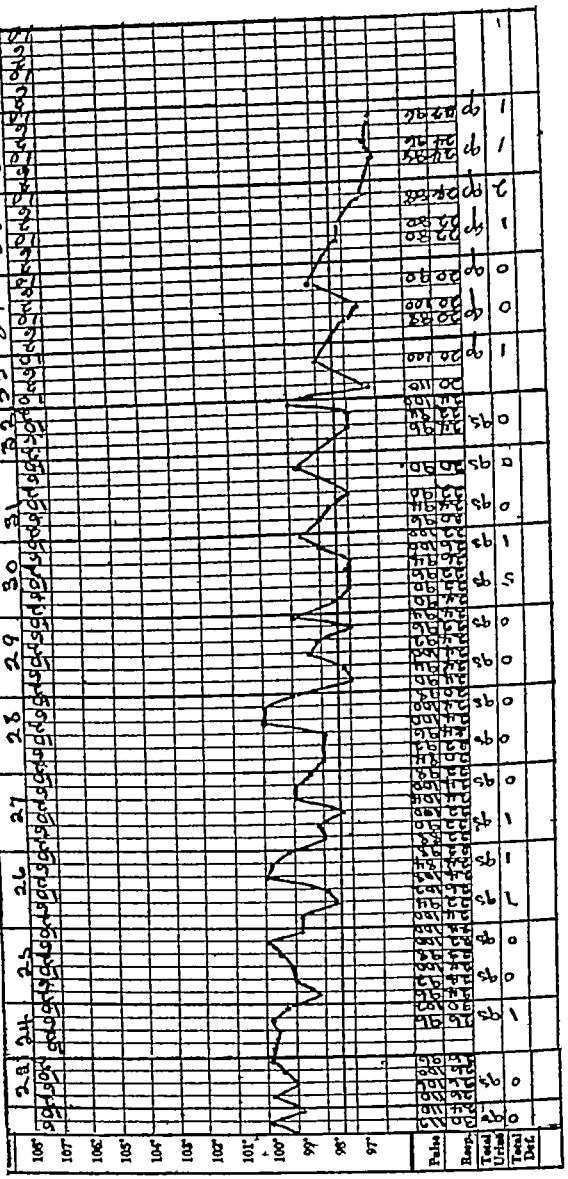

Fig 260 —Temperature chart of Case I

Laboratory Examinations —Sputum negative Wassermann negative Urine shows a heavy trace of albumin, and micro-

scopic examination showed numerous hyaline casts, leukocytes, and pus-cells

Blood —Total leukocytes, 10,800 Differential count normal.

Gastric Contents —One hour after Ewald meal show free HCl 22, total acidity 30, no lactic acid, no occult blood. Microscopic examination negative

Temperature —Examination shows a temperature curve which is septic in character, in that in each twenty-four hours it reaches almost to the normal, and within a few hours there is a rise of three or four degrees

The diagnosis of tuberculosis of the peritoneum was based upon the patient's being a young adult negro, with a disease of rapid onset, ascites with tenderness of the abdomen, discovery of masses of thickened peritoneum and mesentery, and the peculiar temperature curve Because of the temperature curve we could not avail ourselves of the tuberculin test, unless, perhaps, we had relied upon a focal reaction, i e , increase of pain and tenderness in the abdomen, and not upon a rise of temperature.

The case was referred to the surgeon, with a request for exploratory laparotomy The following report was returned

Median incision under local anesthesia Peritoneum exposed, shows much thickening Incised, escape of considerable straw colored fluid Examination of intestines shows walls thickened and hyperemic, and covered with tubercles varying in size from a pin head to a match head.

CASE II —G S Negro Female Age forty three

Chief Complaint —Enlargement of abdomen, pain, and tenderness

Present Illness —Began six weeks ago with nausea, vomiting and pain in abdomen For last four weeks confined to bed because of enlargement of abdomen and pain

Previous history negative. *Family history* negative

Physical Examination.—*General Appearance* —Well developed and well nourished *Eyes* Sclera and conjunctiva slightly jaundiced. Slight hyperemia of vessels *Tongue* clear *Teeth* in fair condition

Heart—Apex-beat in fifth interspace, 9 cm from median line Heart sounds clear Pulse rapid, 90 per minute Regular and full volume

Lungs—Negative throughout. Respiration is slightly increased

Abdomen—Distended, 89 cm in circumference at umbilicus Dome shaped Tympany about umbilicus, dulness in flanks Dulness changing with position Fluctuant wave distinct No masses to be felt, but abdomen distinctly tender all over upon palpation or percussion

Liver and Spleen—Not palpable Skin dry and harsh.

Joints—Right knee flexed, impossible to extend Other joints normal

Lymph-glands not enlarged Temperature septic in character

Laboratory Examination.—*Urine* negative

Blood—Leukocytes 5400 Hemoglobin 80 per cent. Differential count normal No malarial parasites Wassermann negative

Pelvic examination showed mass, irregular in shape, firm, nodular, size of fetal head

The diagnosis in this case was based upon the acute onset of the ascites, rise of temperature, and tenderness of the abdomen As in the case previously reported, we could not avail ourselves of the tuberculin test because of the temperature, and we did not care to produce a focal reaction because of the existing discomfort of the patient

The patient was referred to the surgeon for exploratory laparotomy, and the following report was returned

General Anesthetic—Abdomen contains considerable straw-colored fluid Peritoneum, intestines, and uterus covered with small pearly tubercles Uterus contains large fibroid

In the study of tuberculosis of the peritoneum we are aided very much by the knowledge of the predisposition of the negro race to all varieties of tuberculosis I think an analysis of our cases for the past five years would show full 75 per cent of the cases have been in the negro race

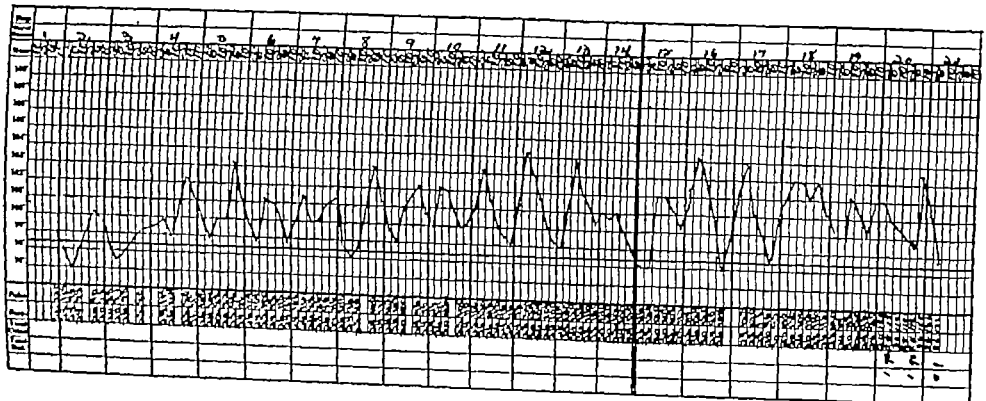

Fig. 261 —Temperature chart of Case II

We should in all of these cases pay especial attention to the occurrence of local tuberculosis elsewhere in the body in deciding upon the presence or absence of the disease in the peritoneum Quite frequently there is a history of glandular tuberculosis, or masses in the neck, as negroes usually call them, or a history of pleurisy, or a disease of the hip-joint

In the case of the female we should never overlook the fact that the fallopian tubes are frequently the starting-point of a peritoneal tuberculosis, and where there is any doubt, a careful pelvic examination by a competent gynecologist might be the means of clearing the diagnosis

Too much stress cannot be laid upon the study of the fluid obtained through the aspirating syringe, or the study of all of the fluid obtained after a paracentesis A very difficult and tedious process is the centrifugation of quantities of the fluid, and staining the sediment by the antiformin method, for tubercle bacilli While this measure is difficult and time-consuming it is possible for tubercle bacilli to be demonstrated

A study of the cells in the centrifugate will usually show a relative increase of lymphocytes, and while very suggestive of tuberculosis, absolute reliance cannot be placed upon it as a diagnostic sign because the same findings may occur in cancer or syphilis of the peritoneum

Attempts to cultivate the tubercle bacillus from the fluid are difficult and too slow to be of any value as a quick diagnostic sign

Inoculation of small quantities of the fluid into the peritoneal cavities of guinea-pigs is a procedure of great value, but objected to chiefly because of the length of time required to produce the disease in the lymphatic glands of the animals Quite recently it has been suggested that the susceptibility of guinea-pigs could be increased, and the length of time for the development of tuberculosis in them much shortened by exposure of healthy animals, before inoculation, several times to the Roentgen rays

As was mentioned in the history of both cases, because of the fever that usually prevails, complete reliance cannot be placed upon the subcutaneous test with old tuberculin Usually, how-

ever, when the proper dose is given there is coincident with the rise of temperature, a focal reaction which is indicated by increase of pain and tenderness of the abdomen, usually accompanied with nausea and vomiting The focal reaction is of great value in many of the cases. There is some doubt as to its absolute harmlessness, but it may be necessary as a final resort to clear up cases which are difficult to diagnose

SYPHILITIC FEVER

In spite of the fact that attention has been called to the occurrence of fever of long duration during the secondary and tertiary stages of syphilis since 1732, and quite recently in the articles of Janeway, Edwards, Herman, and Riesman, it is not unusual for the disease to be overlooked, and for the cases to be treated for long periods under the mistake that they are typhoid fever, malaria, or tuberculosis

This mistake would occur less frequently if we would remember the great prevalence of latent syphilis According to Symmers and Whitney, in an analysis of several thousand cases, during life and at autopsy, which were studied at the medical clinics in two large cities, over 6 per cent. showed unmistakable signs of syphilis The recognition of latent or visceral syphilis will not be overlooked with anything like the frequency that it has been in the past, since the adoption of a routine Wassermann test on all patients entering the dispensary services or hospitals, as is now the rule in most large cities

The fever that prevails in these cases usually assumes the type of a septic or remittent temperature, occurring either over long periods, in some cases for several months, without a decline, or occurring for several weeks at a time with intervals of normal temperature, for a few weeks, with many recurrences In the majority of cases the rise of temperature is associated with chills and sweats, and in some cases the chill, fever, and sweat occur at regular intervals, markedly resembling the periodicity of malaria.

In spite of the prolonged fever, as I have observed in two recent cases, the general condition of the patient continues surprisingly good There is nothing like the emaciation or weakness you would expect in a case of tuberculosis, and the chief and only complaint is the fever, which they are powerless to control

In most of the cases syphilis has prevailed as a visceral manifestation, and the pyrexia has resulted from the absorption of

toxins that have been brought about by the breaking down and healing of minute foci or even a gumma in the liver In other cases there has existed a hepatitis, and in one case reported by Edwards there was an undoubted pylephlebitis

While the liver is the organ most often responsible for the prolonged fever of syphilis, it would be possible for the pyrexia to occur as a result of minute gummata or infiltrations in the spleen, lymph-glands, and even in the gross nervous structures

The diagnosis is usually made from the fact that the cases do not respond to treatment for ordinary conditions that commonly produce prolonged temperatures For example, in the cases reported, the first was mistaken for paratyphoid fever, and the second for malaria These cases are usually kept under observation so long that they wear out themselves and the attendant, and finally are discovered through the means of the Wassermann test, or by the rapid and complete recovery under antisyphilitic treatment

CASE I —Male White Age twenty-three Occupation laborer

Chief Complaint —Chilly sensations, fever, headache, pain in bones, general malaise

Present Illness —Began February 13, 1917, with aching in limbs and back Chills occurred at frequent intervals, followed by fever and an occasional sweat.

Previous History —Twenty or thirty cigarettes a day Moderate drinker of beer and whisky Denies venereal diseases No previous illnesses of any description

Family history negative

Physical Examination.—Well developed and well nourished Eyes normal, react to light and distance Mucous membranes normal Tongue heavily coated on edge No tremor Teeth in fair condition Gums normal Throat Slight soreness and hyperemia of tonsils and pharynx

Heart —Apex-beat in fifth interspace, 3½ inches from median line Heart sounds clear throughout

Lungs —Resonant, and normal vesicular breathing without any adventitious sounds, throughout both lungs

Abdomen.—Distended and slightly tympanitic. A little soreness in right iliac region, a little gurgling upon deep palpation.

Liver.—Not enlarged to palpation. Percussion difficult because of distention of abdomen.

Spleen.—Enlarged, three fingerbreadths below costal margin.

Kidneys.—Not tender or palpable. Skin hot and dry Bones normal. Joints normal. Reflexes normal. Sexual organs negative. Temperature 102 3° F Pulse 96, regular and moderately full volume.

Laboratory Examination.—Urine examined at frequent intervals showed an average specific gravity of 1025, and occasional trace of albumin, never any sugar Microscopic examination showed no casts, a few pus-cells, and bacteria. Diazo test made once was negative.

Blood.—Several leukocyte counts were made. The highest recorded was 13,600, the lowest, 8140 The differential count usually showed a normal proportion of the various types of leukocytes. The blood was specially stained for the malarial parasite, with both the ordinary smear and with thick films, with negative report for plasmodia. A blood-culture made twelve days after entrance and carefully incubated showed no growth. Widal tests with the typhoid bacillus were made at intervals of four days for the first six weeks, and in each instance a negative report was returned April 24th a Widal was made with an undetermined organism supposed to be one of the paratyphoid organisms, and a positive reaction was reported There is, however, no note made of the dilution or of the time limit.

Sputum.—Cough was present, and upon several occasions the patient expectorated sputum. Careful examination was made for tubercle bacilli, but upon five different occasions the result was negative

At this stage of the man's illness the condition was still considered an atypical typhoid fever, but there was a very strong conviction that it might be a case of miliary tuberculosis. Accordingly, an ophthalmologist was asked to examine the eye-grounds. He submitted the following report March 3, 1917 Eye-grounds show an elevation on the central vein in disk of

right eye suggestive of a tubercle This information apparently confirmed our suspicions, and we redoubled our efforts to find a focus, or to find signs of the disease in other organs We were also prepared for an early exit of the patient

In spite of many physical examinations we could never find anything definite in the lungs or other viscera The patient continued in the same way, with the septic temperature, but without any other definite symptoms

During the course of the illness the patient was repeatedly questioned as to the existence of syphilis, but he always strenuously denied it Furthermore, there were no signs of the effects of the disease upon the skin, bones, or glands In desperation to arrive at a diagnosis, a Wassermann test was made on April 24th, and a 4 plus reaction was the report from the laboratory Treatment with small intravenous doses of salvarsan, at weekly intervals, was begun at once Improvement in every way was noticeable after the first dose, and within ten days the patient was afebrile He was in a short time dismissed from the hospital with a complete and uneventful recovery

Close scrutiny of the temperature chart will show it is unlike the normal temperature chart of a typhoid or a paratyphoid fever, and that it conforms to the type of septic temperature. At many places throughout the course the temperature has declined to 99° F , but within a few hours it will be seen to have risen to 102° or 103° F A very confusing feature of the case is the fact that a positive Widal reaction was obtained two months after the beginning of the illness, with a strain of the paratyphoid bacillus I did not see the results, and personally doubt its genuineness

At any rate, the patient presented an undoubted 4 plus Wassermann, and did not improve in the slightest way until he was given salvarsan, with the result of a rapid and complete recovery

Case II —J L White Male Age fifty-nine Railroad man by occupation

Chief Complaint —Chills and fever

Previous History —Usual diseases of childhood No serious

illnesses of any consequence. Syphilis at thirty years of age Treated by usual methods at that time for three years From twenty to thirty years of age drank moderately of alcoholic liquors Since thirty years of age total abstainer Moderate smoker Ten years ago injured in railroad accident, concussion of brain, which kept him away from work for eleven months

Present Illness—Began November, 1916, with what was supposed to be nervous breakdown from overwork Had severe pain in back, malaise, and physical exhaustion Kept away from work forty five days Returned in fair shape, but still not up to normal condition in weight and strength April 18, 1917, began to have chills and fever Paroxysms occurred every second or third day with considerable regularity In beginning chills were dumb, simply manifesting themselves as cold spells, not accompanied by shaking Soon after chilly feeling temperature would rise to 102° or 103° F , followed by a sweat Between paroxysms patient felt entirely normal.

These paroxysms of chills, fever, and sweats continued for one month Blood was examined many times for the malarial parasite, always with a negative result. In spite of negative findings patient was given large doses of quinin by mouth and intramuscularly without any effect whatsoever

In July the paroxysms began to recur, the cold spell usually coming on in the afternoon, followed by a rise of temperature sometimes to 104° F , usually followed by a light sweat. These paroxysms markedly simulated malaria, and even though repeated examinations of the blood, by means of ordinary and thick films, with reliable stains, failed to show anything, it was hard to convince one that the condition was not malaria. The patient continued in the same condition until November, 1917, when he consulted the writer

Physical Examination.—White man, well developed, fairly well nourished

Eyes—Pupils equal, react to light and distance Conjunctivæ slightly bile-tinged Mucous membranes normal Teeth Considerable crown and bridge work Gums show severe pyorrhea. Throat and nose normal

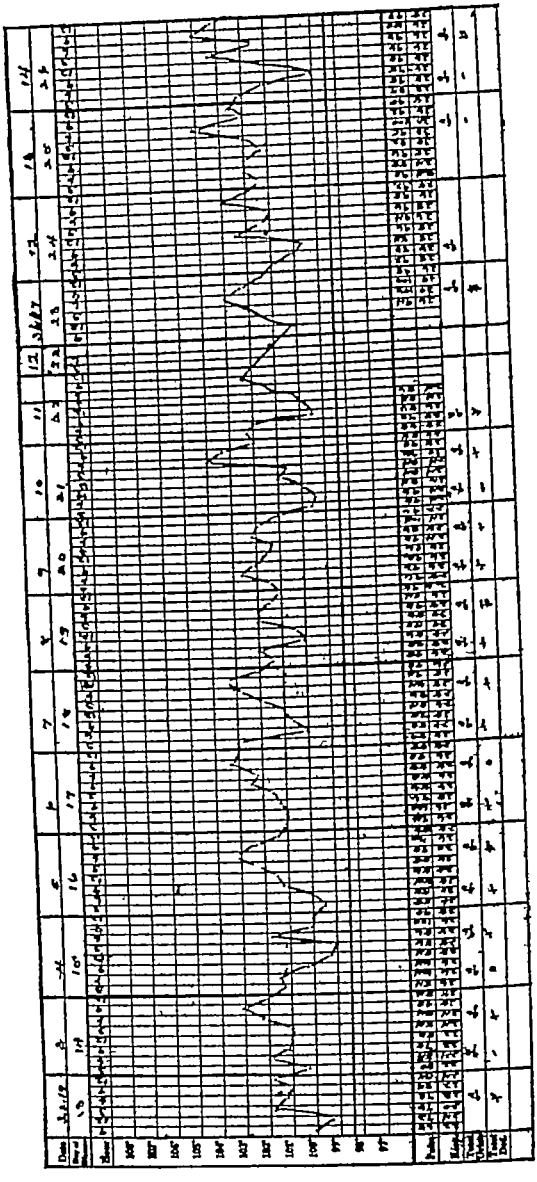

Fig 262 —Temperature chart of Case II.

Heart —Apex-beat in fifth interspace, 9 cm from median line Second sound in aortic area slightly accentuated Heart sounds clear elsewhere

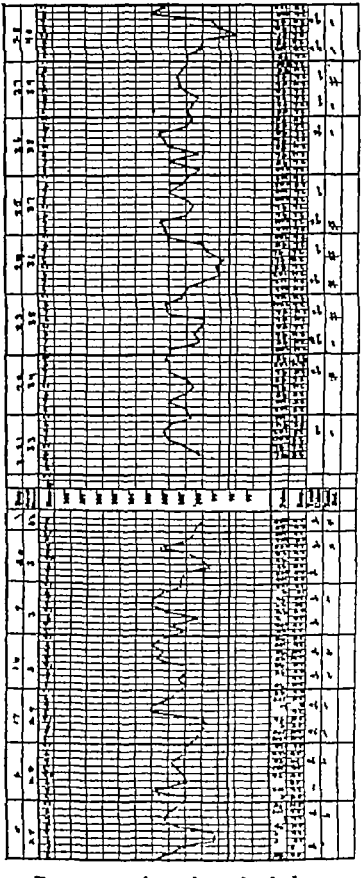

Fig. 263.—Temperature chart of Case II (continued)

Lungs—Resonance throughout both lungs anteriorly and posteriorly Vesicular breathing throughout both lungs

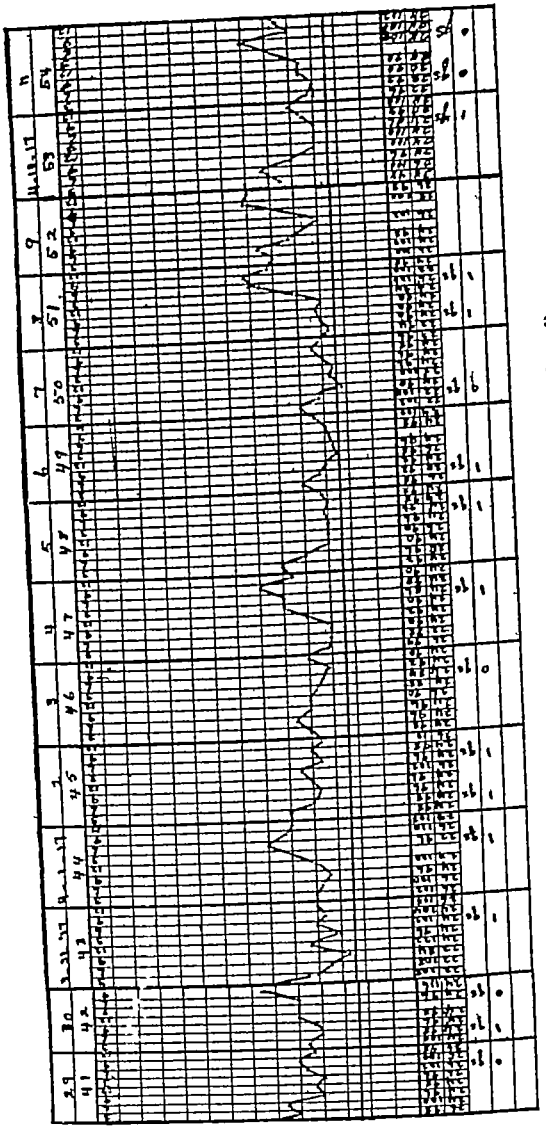

Fig 264.—Temperature chart of Case II (continued)

Abdomen—Spleen not palpable Liver enlarged Upper border normal. Lower border crosses midabdominal line at least 3 cm below midpoint between base of ensiform and umbilicus.

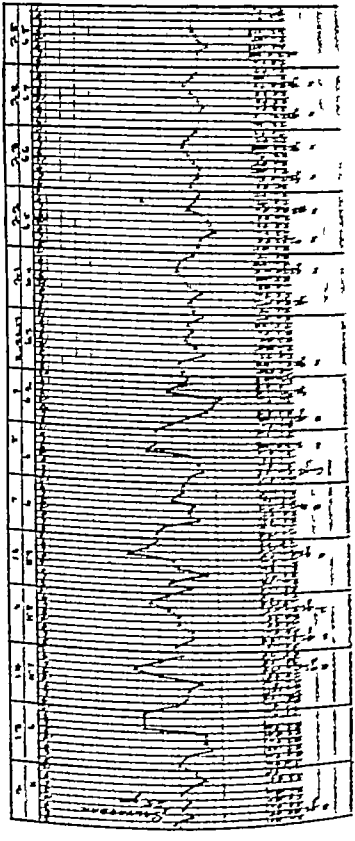

Fig. 265.—Temperature chart of Case II (continued).

Liver slightly tender to forcible percussion and deep palpation. Abdomen not distended nor tender upon palpation elsewhere. *Reflexes*—Patellar active. Gait normal. Joints and bones

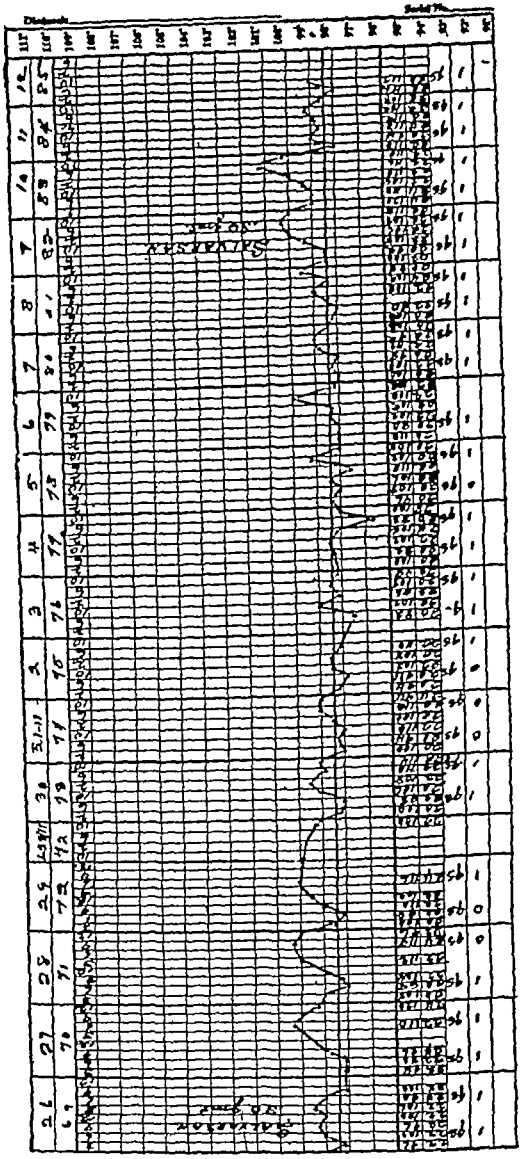

Fig 266 —Temperature chart of Case II (concluded)

entirely negative Glands not palpable Blood-pressure, systolic 140, diastolic 80

Roentgenogram of chest showed heart normal in size Aorta normal. Lungs and pleura clear

Laboratory Reports —Urine examined many times Report December 12, 1917 Acid in reaction. Specific gravity, 1014 Albumin, trace. Glucose, negative Indican, moderate trace Bile pigment, negative. Diazo, negative. Russo, negative. Microscopic examination shows moderate sediment containing few epithelial cells from lower genito-urinary tract, a few hyaline and pale granular casts

Blood always negative for plasmodia. Count made December 12, 1917 Erythrocytes, 4,300,000, hemoglobin, 70 per cent. (Dare), leukocytes, 12,800 Differential count Polymorphonuclears, 77 per cent., transitionals, 5 per cent., lymphocytes, 10 per cent., large mononuclears, 8 per cent., eosinophils, 0, mast cells, 0 Wassermann negative Widal test with typhoid bacillus, dilution 1 50, time limit one hour, showed no agglutination. Test repeated twice, with negative results

Gastric Analysis (Dec. 20, 1917) —Ewald test meal Contents removed in one hour

Amount, 48 c.c.
Reaction acid.
Color normal.
Free HCl, 30
Total acidity, 40
Combined HCl, 10
Organic acids, 0
Lactic acid, negative.
Occult blood, negative.
Microscopic examination, negative

Roentgenogram after barium meal in ten minutes showed a normal Bishop's cap In one hour no filling defects, normal peristaltic waves Stomach cow horn shaped, in normal position. Six hours, stomach entirely empty Eight hours, barium entering cecum and ascending colon

In view of the fact that the patient had a severe pyorrhea, a dentist was called into consultation Roentgenograms were made of the teeth, and several granulomata were found at the

roots of the molars The dentist advised that all the teeth should be extracted because of the severe pyorrhea and several granulomata He assured the patient that the temperature was due to the septic condition of the mouth December 20th, under gas anesthesia, all of the teeth were extracted The gums were treated antiseptically, and healed in due time, without the slightest influence upon the temperature, the paroxysm occurring regularly every second or third day, just as it had been doing for the past two months

In January the patient was seen in consultation with a surgeon A careful examination of the patient's body was made in the hope that a focus of pus could be discovered Special attention was paid to the prostate gland and seminal vessels No evidence at all of any trouble was discovered, except slight prostatic enlargement which usually occurs in a man of that age At the surgeon's request the patient was observed by him for several days Finally, it was decided by the surgeon that the cause of the temperature was either about the liver or in the gall-bladder This seemed incredible in view of the fact that the patient had never had gallstone colic, had never had dysentery, and at the time of the examination there was no sign of jaundice Acceding to the advice of the surgeon an exploration of the liver and gall-bladder was performed on January 12, 1918

Careful examination of the upper and under sides of the right and left lobes of the liver, and exploration with a long needle in several directions failed to reveal an abscess or pus The surgeon carefully examined both kidneys, the spleen, and all of the other abdominal viscera, with negative findings The gall-bladder was opened and showed normal walls, normal bile, no stones, and no signs of infection in the bladder or in the bile-ducts A drainage-tube was inserted in the gall-bladder and the wound closed The patient made the usual surgical convalescence, without any influence upon the temperature During the surgeon's observation of the patient he was, without my knowledge, given daily for eight days 30 grains of quinin intramuscularly, without any result other than a painful abscess in the gluteal muscle

February 10th patient was seen in consultation with a genito-

urinary surgeon A blood-culture was advised This was made and incubated for seventy-two hours, but showed no growth at all A cystoscopy was performed The bladder was found negative No stones Both ureters patulous Urine obtained by catheter from both kidneys collected in sterile tubes and inoculated upon suitable media, incubated for seventy-two hours, showed no growth

During the entire course of his illness the patient frequently called attention to the fact that years ago he undoubtedly had syphilis, and repeatedly asked if it could be possible that the old trouble was recurring As a result of these inquiries two Wassermann tests were made prior to November Since that time several Wassermann tests according to the old antisheep hemolytic system with beef heart antigen, were personally made by me, each time without even a doubtful reaction

In the face of these repeated negative Wassermann reactions it was only reasonable to assume that syphilis was not a causative factor, especially as the patient did not show a sign of the disease in his skin, joints, or reflexes

The patient was fast becoming very restless over our utter inability to control the fever, and in sheer desperation before advising a change of climate (the last resort) I suggested that he allow me to give him an intravenous dose of salvarsan, with the idea of making a provocative Wassermann test, if not directly benefiting him. On February 12th, diarsenol, 0 4 gm in 250 c.c. of saline, was given intravenously In forty-eight hours the blood was drawn for a Wassermann Careful examination showed a doubtful reaction.

In the meantime the patient's temperature had become normal, strength and appetite returned, and there was such a marked and noticeable improvement that he urged me to repeat the dose as soon as it was safe Since that time at weekly intervals the patient has had four doses of diarsenol, his temperature has been normal, he has gained a great deal in strength, and has gained in weight 15 to 20 pounds

I think there cannot be any doubt of the part that syphilis played in the production of fever in these two cases Case I

howed a 4 plus Wassermann reaction, presenting a remittent ype of temperature, simulating an atypical typhoid fever, not esponding to the usual laboratory tests for typhoid fever, show- ng no effort at all to run a normal course, as a typhoid fever, and nly responding, and then rapidly, to antisyphilitic treatment.

In Case II the evidence is just as complete While the Vassermann reaction can usually be relied upon when it is posi ive, it is known by every laboratory man that it means nothing vhen it is negative, and no one would dare say that an individual lid not have syphilis because one or even several Wassermann ests were negative

The irregularity of fever in the case, and the fact that during a ew months in 1917 there was a remission, can be explained upon he theory that gummata or foci in the liver or other abdominal iscera had healed during the time, and others had appeared and roken down later on and had produced the temperature.

Cases of this character, with the successive healing and recur ences of gummata with irregular temperature, have been re- orted by many observers Especial attention has been called o this characteristic of the disease by Billings.

[illegible]

[illegible]

[illegible]

[illegible] and the [illegible] present [illegible] to the [illegible] [illegible] was [illegible] The ambulance [illegible] [illegible] This suggests the [illegible] of all unknown [illegible] in the police station.

[illegible] The [illegible] probably an Italian. He has [illegible] which suggest that [illegible] may have recently been [illegible] of the United States. He is in a state of [illegible] to consciousness by shaking [illegible] caused by pulling the hair. The pupils [illegible] and the patellar reflexes are below normal, but present.

[1] [illegible] examination does not reveal any thing ab[illegible] [illegible] The liver is enlarged about two finger[illegible] below the costal margin. The spleen is enlarged about [illegible] fingerbreadths below the costal margin and is very hard

There is no tympanites There is no apparent edema of body or extremities

We should think of nephritis or diabetes in the presence of coma of unknown cause, and this calls for an examination of the urine A specimen was promptly obtained by catheterization, and the report is that it contains a trace of albumin, but no casts. There is no sugar present This examination eliminates nephritis and diabetes as possible causes of the present condition

The patient's temperature by rectum is 105 2° F As soon as he was brought in the intern, Dr ———, very properly collected a blood specimen for microscopic examination He has already reported that there are large numbers of estivo-autumnal plasmodia present He says that apparently about one-third of all the erythrocytes contain parasites There is no apparent leukocytosis, but no count has been made

The diagnosis of comatose malaria of the most severe form is now very plain and the patient's life is in great danger Whether he recovers or not depends very largely upon getting a dose of quinin into his blood-stream with the least possible delay We will therefore proceed to give him a dose of quinin intravenously at once After doing that we will discuss the case further

We have a 20-c c all-glass syringe with a No 24 needle, direct connection, all sterile We drop two 5-grain tablets of quinin bimuriate into the barrel and put the piston back in place We now draw up into the syringe a few cubic centimeters of sterile water and with a little shaking of the syringe it is quickly dissolved Next we expel a little air that was present and draw up more water until the syringe contains about 20 c.c. It is ready for administration

While I was talking to you and preparing the quinin solution my assistant has, with the best of intention, very awkwardly constricted and prepared the arm Note that he tied a bandage around the arm to distend the vein Please note here on the other arm how perfectly satisfactorily the vein can be distended by gripping with one hand above the elbow The pressure is under better control, it can be released more easily and is more satisfactory in every way

In the case the hope of saving the patient's life depended upon getting sufficient quinin into his blood-stream to kill the malarial parasites present there. The patient being unconscious, it would have been difficult at least to get him to swallow quinin in any form, and if swallowed it would be very doubtful when enough reach the blood-stream. When taken into a normal stomach quinin appears in the urine in about two hours and the maximum amount is eliminated about the eighteenth hour. For this patient to have to wait several hours for quinin might prove fatal. On the other hand, when we introduced the quinin directly into the blood-stream its effect upon the parasites was certain and began in full force at once.

Experiments show that when quinin is given intravenously the rate of absorption and elimination is about the same as when it is given by mouth, provided it is properly diluted. If on the other hand, the solution given is too concentrated or is injected into the subcutaneous cellular tissue the absorption is much

slower In instances where it produces necrosis of tissue and abscess (and these instances occur quite frequently) the quinin is never absorbed Even where severe inflammation of the tissues occurs (and this is very frequent) the quinin only reaches the blood-stream slowly over a period of several days

Thus you see that it would be several hours or even several days before quinin given intramuscularly would reach the blood stream, and the patient might die of malaria with enough quinin in his tissues to save him if it had been put in his blood. When we give it intravenously, on the other hand, all is ac complished that may be accomplished by quinin.

It must not be expected that quinin given intravenously will save every case of comatose malaria, for in many instances the damage already done will be destructive of life in spite of the treatment Again, it must be understood that quinin only kills those parasites that are in the circulating blood, but it does not affect those lodged in the capillaries as long as they remain there.

The life cycle of the estivo-autumnal malaria parasite is thirty six to forty-eight hours During the first eight to twelve hours of this period the parasite is small and is to be found in the circulat ing blood After this time, when the parasite gets larger, past the ring stage, it can no longer pass through the capillaries, but lodges in them, where it remains during the balance of the life cycle Capillary loops are obstructed by the parasites lodged in them and, of course, no blood flows through such obstructed capillaries Whenever sufficient of the capillaries of the brain are obstructed in this way there is much anemia of the brain and the patient becomes comatose We call it comatose malaria. It would not matter how much quinin there was in the circulating blood, none of it would reach those parasites in these closed capillaries After a parasite segments in a capillary the young parasites are swept out into the blood-stream If quinin is present in the blood in sufficient concentration, they are killed Young parasites are continuously appearing in the blood-stream, especially in such severe infections as in this case It therefore is desirable to have quinin in the blood-stream continuously to kill them as they appear

Whenever a dose of quinin is given by mouth or intramuscularly it reaches the blood-stream slowly, but it is also slowly eliminated. On the other hand, when given intravenously it is much more rapidly eliminated, and in a few hours following a given dose the blood would contain only a small amount of quinin. It therefore is necessary to repeat the quinin every few hours, and in urgent cases as often as every two or three hours In this case we will give another dose of 7½ grains in about two hours After that time he will be given a dose not exceeding 7½ grains about every six hours until he is able to take quinin by mouth. If he recovers, it is most likely he will be able to take it in this way within twenty four hours from now Just as soon as he can take quinin by mouth the intravenous quinin will be stopped When ever quinin has been given intravenously quinin by mouth is frequently neglected This should never be done.

Please let me call your attention to the doses I employ Here we have perhaps the most urgent condition we will ever see in malaria, and I have employed what I consider the largest dose ever demanded or justified Ten grains of quinin will kill just as many of the parasites in the circulating blood as any larger dose will, and there is no danger of doing the patient great harm. Much larger doses, even 20 or 30 grains, are dangerous, and in some instances may be destructive of life. Of course, with a patient in such extreme condition as this one, if he should die within a few hours as a result of the toxic effect of quinin, we would not be likely to recognize the true cause of death In a number of instances in my experience comatose malaria cases have been given larger and larger doses of quinin intravenously until they finally died of quinin poisoning At the time of death there would be few if any parasites in the circulating blood and the autopsy would fail to show sufficient malaria parasites in the tissues to explain the death I would hesitate to say what percentage of all the deaths in comatose malaria in my experience I think were due to the toxic effect of excessive doses of quinin given intravenously especially

As soon as this patient can be given medicine by mouth the specific treatment from that time on will be the same as it would

be for any other case of severe malaria That would be 10 grains of ordinary sulphate of quinin three times each day for a period of three or four days, and then a dose of 10 grains every night just before retiring for a period of eight weeks At the end of this period of systematic treatment the patient should be free of malaria parasites and would not be in danger of suffering a relapse

NOTE —[The subsequent history in this case is that the patient improved somewhat more rapidly than was expected when he was first seen in the clinic. Within three hours the coma had almost entirely cleared up The patient talked fairly intelligently at times, but seemed more or less dazed, and would quickly drop back to sleep when let alone The condition finally cleared up in about twenty-four hours The temperature was recorded normal just forty hours from the time he was given the first dose of quinin intravenously

Three hours after the dose of quinin was given in the clinic he was given another dose of 7½ grains intravenously, and about one hour after that he took 10 grains of quinin sulphate by mouth. He was not given any more quinin intravenously, but took 10 grains every eight hours for four days, and after that 10 grains each day His improvement was rapid and uneventful. On the ninth day after admission he left the hospital, contrary to our advice, to join his ship which was leaving port At that time he was still quite anemic, hemoglobin 55 per cent , and his spleen still enlarged, though smaller than upon admission]

In all cases of acute malaria, a preliminary purgative of one kind or another is beneficial and should be administered There is much difference of opinion as to whether a simple purgative, such as the salines, is best or whether a dose of calomel followed by a simple purgative is best I am not sure that the calomel has any special advantage, but confess that I prefer and use it in all acutely ill malaria cases One thing I am sure of is that whatever purgative may be given, it is not proper to wait for its action before beginning the quinin The purgative may be helpful, but quinin (and other cinchona alkaloids) is the only specific remedy known for malaria Nothing should stand in the way of starting

its use as soon as the diagnosis of malaria has been made. The time to start quinin in any case of malaria is when the diagnosis is made

Only recently an instance came to my knowledge where waiting for the action of a purgative resulted in the death of a patient who should have been saved. A child six years of age had a very severe chill and high fever This was the third chill and fever coming at the same time of day on each of three suc cessive days A physician was called and he promptly made a definite diagnosis of malaria and recognized the severity of the case. He prescribed some calomel to be taken that night to be followed by a saline the next morning, and advised that quinin be started after the purgatives acted off It happened that the purgatives did not act during the next day after his visit, and therefore the quinin was not given.

In the afternoon another very severe chill occurred, followed by convulsions and later coma. The physician was again called and then started giving quinin hypodermically During the following six hours he gave this six year-old child 60 grains of quinin dihydrochlorid hypodermically or intramuscularly This would correspond to about 200 grains for an adult. The child died the next day, never having come out of the coma It is impossible to know whether malaria or the quinin was the cause of death, but it is reasonably certain that if proper quinin treatment had been started at once upon the first visit the malaria would not have caused death nor would there have been any excuse to administer such large doses of quinin. That amount of quinin, if absorbed, would be likely to cause death If it was not absorbed, as was probably the case, the patient died with many times enough quinin in his tissues to have saved him if it had been given earlier and properly Do not wait for the action of an antipyretic or a purgative before starting proper quinin treatment in malaria. Begin the quinin at once in all cases

The next case for our consideration is reported to be a case of malaria in which quinin given by mouth and also hypodermically has failed to relieve the clinical symptoms of the disease The

patient was brought to the hospital by Dr ——— from Slidell, Louisiana, arriving last night The doctor has kindly waited over and has given us a complete history of the case

The patient, Mr E A , is thirty-four years of age, and is a ship carpenter He claims not to have been sick in any way in twenty-one years until the present time He lived in Cleveland, Ohio, all his life until he came to Slidell, La , about eight months ago He drinks beer occasionally

About one month ago he had a chill followed by fever The physician called in that evening decided it was malaria and gave him some calomel and quinin He took the quinin for about a week according to his physician's instructions and has remained well until the present attack, which began just three weeks after the previous attack

Eight days ago, while working in the shipyard, he was taken with a chill and vomited a few times This was promptly followed by severe headache, backache, and considerable fever, which he thought was the same trouble that he previously had Instead of calling a physician that day he decided to take some "Black Draught" with which his roommate assured him he had stopped the malaria on himself many times He did not return to work the next day, though he felt fairly well, but did return the following day At about the same time in the afternoon he had another attack of chill followed by fever, and this time called in the same physician

The doctor felt confident that this was a relapse of the malaria he formerly had, but decided that before putting the patient on quinin again he would have the diagnosis confirmed by microscopic examination of the blood

He sent a blood specimen to the State Board of Health Laboratory and received a report the next afternoon that estivo-autumnal malaria plasmodia were present, both rings and crescents The patient was started taking quinin that night, 5 grains every six hours The next day he had another chill and fever at about the same time of day the previous paroxysms began, and quite as severe as they were

Disappointed at the failure of the quinin given to prevent the

chill, the doctor decided to try giving it hypodermically A hypodermic of 10 grains of quinin bimuriate was given that night following the chill, and a similar dose night and morning for three successive days Both the physician and patient were delighted with what were considered good results from the hypodermic administration of quinin, and no doubt they were impressed with the apparent superiority of this method of administration over the older method of administration by mouth. On the fourth day, however, the patient felt dull, languid, had a headache, and about noon had a slight chill. His temperature was 102.2° F when seen by his physician at 2 o'clock It was then decided to bring him to the hospital and learn what explanation we might offer for the failure in treatment, and especially what method of treatment would be advised Dr ——— is present and the patient is before us now

The patient appears well nourished and not very sick. You will note, however, the flushed cheeks and eyes rather injected You have noticed, no doubt, that since we have had him in the amphitheater he has been rather restless, turning on his cot frequently, as if trying to get comfortable. This may be due to nervous temperament and unimportant, or, what is more likely, it may be due to his fever, which is 101 6° F , and to the condition causing it.

There is nothing noteworthy recognized about the head and neck except the flushed cheeks and injected eyes This is no more marked, however, than may be seen in fever due to a variety of things

Nothing abnormal in the heart and lungs can be made out Pulse 102, regular, rather full Respiration 24 per minute.

The liver cannot be palpated and no liver dulness can be made out below the costal border

Since malaria is especially to be considered, we should give special attention to the spleen There is almost always some enlargement of the spleen in cases of malaria of more than a few days' duration. Yes, this one is distinctly palpable when the patient forces it down with the diaphragm by deep inspiration. With the patient lying on his back and his knees drawn up to

relax the abdominal muscles I can palpate best with my right hand resting on his abdomen and the tips of my fingers directed up nearly to the costal border With my left hand back of the spleen region I lift it forward This tends to shove forward and render an enlarged spleen more easily palpated Now as the patient takes a deep breath the edge of the spleen strikes against my finger-tips and is readily recognized Let us see if we cannot demonstrate it so you can see from your seats When the patient takes a deep breath and forces the spleen against my fingers I press upward against it somewhat beneath the edge. Then I slightly relax the pressure and allow the edge of the spleen to slip suddenly beneath my finger-tips It is distinctly felt and you see the fingers suddenly slip over it

The abdomen is rather flat, the patient having been given a saline last night and an enema this morning Nothing abnormal is made out There is no tenderness

The patient complained of my hurting his arm a moment ago, and now upon examination we find what should have been observed or found out sooner There is an area of purplish redness about 4 inches in diameter around the insertion of the deltoid, which we are informed is the site of the first hypodermic injection of quinin given five days ago It is painful and when compared with the other arm is somewhat swollen Upon deep pressure the center especially feels boggy Here we have a probable explanation for the fever he now has—inflammation and probably necrosis of tissue resulting from the hypodermic injection of quinin

The blood was examined early this morning for malaria plasmodia and none found' Another examination was made about 10 o'clock, with the same result as to malaria There were 15,400 leukocytes per cubic millimeter and the differential leukocyte count showed small mononuclears 16 per cent , large mononuclears 2 per cent , neutrophils 82 per cent , eosinophils none, basophils none A moderate leukocytosis and a definite "septic factor" is present We are justified in concluding that the present condition is due to the effect of a hypodermic of quinin and not to malaria If malaria was the cause of the present fever

we should be able to find malaria plasmodia in the blood and there would be no leukocytosis Simple malaria never causes leukocytosis or the "septic factor" in the differential leukocyte count.

We should emphasize also that this is not a case of malaria in which quinin has failed to control the clinical symptoms of malaria, as it was supposed to be

Very likely the quinin that caused so much local damage was injected rather superficially into the subcutaneous cellular tissue and not deeply into the muscular tissue. It is much less likely to produce necrosis and abscess when put into the muscular tissue. The solution used was also most likely very concentrated The more dilute the solution, the less likely to do damage. I do not think quinin should ever be given hypodermically or intramuscularly, but if there ever occurs an instance when quinin should be so given it should be diluted at least ten times Ten grains of quinin dihydrochlorid should be dissolved in no less than 6 c.c. or 1½ drams of water

As for the treatment in this particular case, we have a condition likely to require surgical attention in a patient also known to have malaria. It will be impossible to say at the present time whether the damaged tissue will break down and abscess or not, but most likely it will. The damage is done by the chemical action of the quinin on the tissue causing inflammation or necrosis, and not to infection, as is often supposed. Unless there is considerable improvement in both the general and local condition shortly, incision, permitting drainage, will be indicated

So far as the malaria is concerned, the patient should be treated as any other patient who had just been relieved of the clinical symptoms of malaria would be treated That treatment is 10 grains of quinin sulphate in capsules every night for a period of eight weeks without intermission He will then be free of malaria parasites and will not relapse again.

The third patient was kindly sent in for our clinic from the Outpatient Department. The diagnosis made is malaria relapse, intermittent malaria

Mr J C McL, age forty-two, a resident of New Orleans for the past twelve years He has been a bill clerk in a wholesale grocery store for the past seven years

His family history has no bearing upon the present trouble except that two younger brothers were having chills and fever at the time he paid a visit of a week to his family in Mississippi, in June, 1915

The only sickness he has had during the past several years has been repeated attacks of chills and fever during the past three years He now comes to the clinic for one of these spells of chills and fever of about one week's duration

He visited his old home near Hattiesburg, Mississippi, in June, 1915 At that time two brothers were having chills and fever In about ten days after returning to New Orleans the patient had a chill one morning about 11 o'clock, followed by headache and fever, which passed off during the night. He felt pretty well the next day, but on the following day another chill came on just forty-eight hours from the onset of the previous one. He treated himself with chill tonic, which broke up the chills after the third one had occurred

In a few weeks he had another attack, having a few chills, each followed by a few hours of fever of the same type as before. This time he consulted a physician, who gave him a prescription and told him to take some quinin

Since that time he has had two or more attacks of one or more chills and fever each every year He has been treated by three different doctors during the time They always stopped the chills, but they would come back Patient says he has taken a "bushel of quinin" and "one doctor put it into" him with a hypodermic needle This was in the fall of 1917, and he thought he was cured, but now he is disappointed that it "has come back on" him again He is quite confident there is no use for him to try quinin any further

During the present illness the patient has had four chills followed by fever, each occurring about 2 P M every other day, the last one occurring yesterday The chills last fifteen minutes to half an hour and the fever not more than six or eight hours

There is much headache and backache during the attack. Patient says he "burns up with fever" Also that the present attacks are just like those he has been having for three years

Upon physical examination nothing abnormal is found. There does not seem to be any enlargement of the spleen The patient, as you see, does not appear to be sick He looks fairly well and there is no anemia apparent

A blood examination made this morning shows tertian malaria plasmodia present and a few stippled erythrocytes. There is no leukocytosis The differential leukocyte count is within the normal limits Hemoglobin by Tallqvist's scale 80 per cent.

The diagnosis is tertian malaria. In view of the history and in view of the fact that he has not been out of New Orleans, where there is practically no transmission of malaria, since 1915, it is reasonably certain that this is a relapse produced by the original infection he got three years ago

Careful inquiry from him indicates that from first to last he has taken quinin during more than three months of the three years since the infection occurred Most of this was taken in very variable doses, however, and for a few days at a time. In one instance he says a doctor gave him two capsules (a very indefinite quantity) of quinin every day for three weeks He has not, therefore, taken quinin in such a way as to be at all certain to get rid of the infection, though, as he says, it always relieved the symptoms for a while.

Our advice is that he take 10 grains of quinin sulphate in capsules three times each day for three successive days Usually if the quinin is started twenty four hours or longer before the next chill is due it will be missed If only started a shorter time before the next chill is due the chill will often come on, no matter how large the dose of quinin After he takes 30 grains of quinin a day for three days, then he should take 10 grains every day (preferably every night before retiring) for a period of eight weeks without intermission At the end of this treatment he will be free from infection and will not relapse again

CONTRIBUTION BY DR. LAWRENCE T ROYSTER

NORFOLK, VA

THE CARE OF THE PREMATURE INFANT[1]

IN the care of the premature infant we are confronted with one of the most difficult and serious problems in medicine, there is no problem, however, which offers a more gratifying reward of success when painstaking, intelligent management is instituted Attention to details is essential to the success of all medical and surgical cases, even the aseptic technic of the operating room is of no more importance than attention to the minutest detail in handling a premature child Under the best conditions the mortality is exceedingly high, but many children can be saved if given proper attention The life of the child depends largely on the degree of prematurity and state of development. For example, a child may be born at seven months weighing more than another born at eight months But a six or six and a half months' fetus weighing 3 pounds or less has, under the best conditions, an exceedingly poor chance Cesarean section in obstetric emergencies has vastly increased the chance of life of the premature from these causes over any form of accouchement forcé.

The main indications in handling premature infants are "the maintaining of normal heat, the nourishment of the infant, and the prevention of infection," therefore, the subject will be discussed along these lines

All newborn infants have the heat regulating center poorly developed Even with normal infants it is unwise to expose them to sudden changes of temperature. It must be remembered that at birth the infant passes abruptly from an environment the temperature of which is 100 degrees to one 15 to 30 degrees lower

[1] Read by invitation before the George Washington Medical Society Washington, D C., April 16, 1918.

This is naturally a shock—one often sufficient to determine the matter of life or death of a very weak, premature infant Hence, every effort should be made to overcome this shock where possible Only too often, not only in house practice, but even in some of our best hospitals, no provision whatever is made for the care of the premature The mother's condition may be so grave that her welfare alone is considered by both physician and nurse, and the infant is neglected sometimes for hours This should not be the case Some capable person should be delegated with the entire care of the newborn patient, and, preferably, if the case is to be turned over to a pediatrician he should be present so as to assume the care of the infant from the moment of its birth. When a premature birth is anticipated, whether normal or operative, a warm blanket covered by a warm sterile sheet or towel should be kept at hand in which to immediately wrap the baby This procedure alone will save many of these little ones

If the infant is born asphyxiated it has a serious handicap, for the premature cannot well stand the usual methods of resuscitation and very little slapping or waving should be permitted, under no circumstances should the child be submerged in cold water All necessary resuscitation should take place in a room sufficiently heated to prevent material radiation The maintaining of body temperature should then be our first care, and this can be done always within 1 degree and often within the limits of $\frac{8}{8}$ degree Because of the poorly developed heat-regulating center already referred to the infant responds very rapidly to its environment It must be understood that the handling of the infant must be done with surgically clean hands on the part of the attendant

The infant should not be bathed at all, but should, on alternate days, be oiled with warm sweet oil The question of incubators is a mooted one Outside of well-regulated hospitals with nurses trained in the handling of premature infants and the management of incubators they are not successful Personally, I do not employ them, instead I have with good success made use of the so-called premature infant's gown or jacket (Figs 268 and 269) This is made of non-absorbent cotton batting, about $\frac{1}{2}$ inch

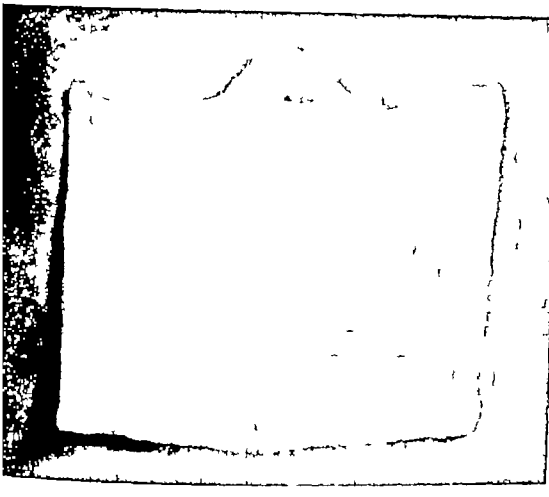

Fig 268.—Premature infant's jacket

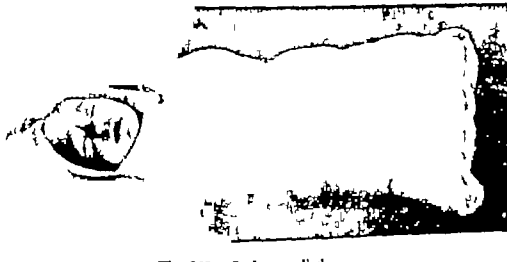

Fig 269.—Jacket applied.

thick and quilted between layers of gauze with a hood attached which covers the head This is made as a one piece garment No clothing whatever is employed As soon as the infant is delivered

it is well oiled and placed immediately in the jacket, which is pinned up the front with safety-pins in a similar manner to a surgical binder The bottom is then turned up and pinned No napkin is used, but in its stead a small pad, 4 or 5 inches square, made of quilted cotton and gauze similar to the jacket, except that for the pads absorbent cotton should be used These pads are changed whenever soiled A hamper clothes basket or bassinet is employed as a receptacle A pillow is placed in the bottom and pressed down in the middle, the baby now enclosed in the jacket is placed therein and covered with several folds of blanket and hot-water bottles or bags placed so as to surround the infant

The infant is not removed from the basket for any purpose save its oiling and weighing every other day, except when strong enough to nurse, and then it is put to the breast in the jacket. The body temperature should be maintained between 98° and 99° F , which can be done readily by means of hot-water bags or bottles or by an electric pad, which may be attached to any socket I am told that these pads require close watching, since they sometimes get out of order and may cause trouble The infant's temperature should be taken at four-hour intervals per rectum, for this purpose the jacket is opened at the bottom and the nurse's hand run up through the jacket to the baby's rectum

In order to aid us in maintaining the temperature another thermometer, such as an ordinary bath thermometer or one enclosed in a glass tube, may be placed between the infant's jacket and its covering blanket So long as this is kept at 90° F we are safe in assuming that the baby's temperature is within the desired limits On alternate days the infant is transferred in its basket to a room such as a bath-room which has been previously heated to as near 90° F as possible Everything being in readiness, the jacket is removed, the baby is quickly weighed and oiled with warm oil, and again wrapped in a clean warm jacket This whole procedure need not occupy more than two or three minutes This method of maintaining heat is quite as successful as the use of an incubator The infant is usually kept in the jacket until its weight is 6 pounds

The second indication, that of nourishment, presents the greatest difficulty Two facts must be constantly borne in mind. First, that the infant is born at a time when its powers of digestion are not sufficiently developed to take the food which is usually supplied to a full term baby, and second, its strength to nurse is so slight that breast or bottle feeding is quite out of the question

Breast milk is, of course, desirable, in fact, in the very delicate is absolutely essential to success Generally the mother's own milk does not appear promptly in the very premature cases, and recourse must be had to the milk of some other woman. In the meantime a strong, lusty baby may be put regularly to her breast for the purpose of stimulation. As a rule, a premature infant cannot digest whole human milk, so in the beginning even this must be diluted one-half and in some instances one fourth with 5 per cent. milk-sugar solution This is gradually increased in strength until human milk of full strength is administered As the child develops, if it becomes necessary to give up human milk we may safely give modifications of cows' milk. In some instances it may be impossible to secure human milk and it may be necessary to use cows' milk from the beginning A number of my most successful efforts have been under such circumstances When such a procedure is necessary the following outline is suggested

The usual initial loss in weight of newborn infants may be in part physiologic, though sometimes it is no doubt due to the fact that the mother's milk does not appear until the third day Dr Fritz Talbot has successfully demonstrated that this initial loss may be prevented in large measure by giving a 5 per cent. solution of milk-sugar from birth. Premature infants can ill afford any initial loss, however small, hence I have adopted the routine of administering a solution of milk-sugar at once This is replaced by some other form of food as soon as possible. I have found whey of inestimable value in these cases This may be made from whole or skimmed milk, according to whether we think the infant can take any fat at all or not. If made from skimmed milk we have a formula which is approximately F0

S4 50 P90 (whey protein) This is usually well borne and will serve to prevent any loss, and even in most instances furnishes sufficient food to cause a considerable and steady gain in weight. However, we cannot expect a gain for a very long time on such a mixture, and so when the child appears to have a fair start we gradually change to a formula of cows' milk This change must be made at first by adding skimmed milk to the whey, thus giving the so-called "split protein," *i e*, whey protein and casein. From this we begin to skim off less and less cream from the milk used to make the whey, until we have whey made from whole milk, which gives a fat percentage of 0 90 When this stage has been reached we change to a formula of cows' milk of approximately F 75 S 5 P1 After this the conduct of the case may safely be pursued along regular lines of increase

The interval of feeding and the quantity taken at each feeding depend entirely on the infant, as is the case in feeding a normal well infant A small premature rarely takes more than ½ ounce at a feeding, and many times 2 drams may be the limit The best interval for quite a while is two hours

The manner of administration varies with the exigencies of the case A strong and but slightly premature infant will take the breast or bottle readily, the further we get away from full term, however, the more difficult the problem becomes The Breck feeder is very useful not only as a feeder but also in teaching the infant how to nurse More frequently we have to begin with a medicine-dropper, and quite often it becomes necessary to resort to gavage where the infant is too weak to nurse To keep the baby alive it is quite obvious that we must supply the nourishment in whatever manner necessary Gavage is not difficult. A small rubber catheter and a glass funnel are all that is needed The tube is passed either through the nose or directly into the esophagus through the mouth During this procedure the infant does not have to be moved from the basket It is very important to keep the food warm while waiting to use it, and the best way to do this is to set the container in a bowl of warm water

The third indication—prevention of infection—is exceedingly important The avenues of infection most usual in these cases

are the mouth, the lungs, and the umbilicus The mouth should be kept as clean as possible. It must be remembered that a baby's mouth is sterile, and if infection occurs it does so from without. Sterile water should be used once each day Absorbent cotton and not gauze should be wrapped about the little finger, dipped in the sterile water, and every part of the mucous membrane of the infant's mouth cleansed thoroughly, but very gently The habit of washing out a baby's mouth at each feeding is pernicious and should be abandoned. The greatest care should be exercised in keeping the nipples clean. The part of the nipple which goes into the mouth should not be touched after boiling I do not keep nipples in solution at all. After a thorough cleansing and boiling they are kept dry in a preserve jar or other covered glass receptacle. At the slightest indication of coryza on the part of either the attendant or the nursing mother masks should be used to prevent the infant's acquiring the coryza and possibly a subsequent pneumonia.

The incidence of pneumonia should be scrupulously guarded against, since this disease causes a very large number of deaths among premature infants Although it is necessary to maintain body temperature as already outlined, the infant must not be kept in a close, stuffy room. The room in which the baby lives should not exceed 72° F at any time, and a free flow of fresh air should be assured In very cold weather in steam heated rooms the heat should be kept in the radiators, but the windows raised enough to secure free circulation Whether radiators or stoves are used, moist air must be assured by a pan of water constantly kept near or at the source of heat.

Cool fresh air does not cause pneumonia, but rather prevents it. Hot, stuffy air is one of the best means of producing it.

Prevention of infection of the cord is easiest accomplished by letting it absolutely alone when the proper initial dressing has been applied until it has come off and has healed

CONTRIBUTION BY DR J ROSS SNYDER

BIRMINGHAM, ALABAMA

A STUDY OF 10 PELLAGRINS IN TWO FAMILIES LIVING IN THE SAME NEIGHBORHOOD[1]

ON the morning of June 20, 1917, a mother carrying a sick child in arms applied at the Children's Hospital for the services of a physician There is no resident physician at this institution, but the mother was urged to await the arrival of the attending physician Although the mother believed her child to be dying, she said she would be compelled to go back home because she had left there other children unattended She refused the offer to admit the child to the hospital Soon after her departure I was informed of the above facts Obtaining the address, which the admitting nurse had fortunately secured, I investigated the case. Some interesting findings were made

The Johnson family consists of father and mother and five children The father is thirty three years of age. The mother is twenty nine years of age The family has been living at the present location since September, 1916 Previous to this time the family had lived in the country The father is a section hand on the railroad His earnings are usually about $50 per month. For several weeks the father has been unable to work steadily because of bowel trouble and sore hands There is no other source of income except the father's wage The neighbors occasionally send in vegetables, such as cabbage greens and radishes The whole family has been sickly for several weeks, the father and several of the children have loose bowels The father and all the children have a breaking out." The mother has no loose bowels, no breaking out, but her mouth is sore and

[1] Read before the Jefferson County Medical Society April 29 1915

her stomach burns She also has headaches and dizzy spells The mother believes the sickness of the family is due to self rising flour, several sacks of which were bought by her husband at a sale The chief articles of diet during the winter months were corn-bread, potatoes, fat meat, molasses, field peas, cabbage, and coffee—on some Sundays pork chops No milk except an occasional can of condensed milk which was bought for the twin babies, but which was never given with regularity, days often intervening between the exhaustion of the can and the ability to purchase another Since the warm weather there has been but little change in the diet except that biscuits from the self-rising flour have taken the place of corn-bread At the time of my visit the sickest child is lying on the mother's lap and being urged to take some underdone biscuit dipped in syrup The other children are seated around the dinner table. The meal consists of cold boiled potatoes, biscuit, and syrup If any animal protein is to form a part of the meal it will be furnished by the accidental ingestion of some of the numerous flies which swarm over the food and over the children's hands and faces The child in the mother's lap is evidently very ill The mother gives his age as three years, but except for length he looks like a marantic infant. The eyes are sunken, the lids semiclosed The lower jaw sags—the mouth is open. The child feebly pushes away the offered food The tongue and mouth are dry The tongue is coated a yellowish brown, except on tip and edges, where it shows red The teeth are sound, but the gums are swollen and red The abdomen is scaphoid The integument of the skin is dry, wrinkled, and loose The child has an erythema over the entire face except for a narrow margin running parallel with the hair-line There is also an erythema on his neck, hands, feet, and legs There is a distinct line of demarcation between the affected and the normal skin The erythema is bilateral and symmetric. The temperature (rectal) is 103⅘° F , the respiration is shallow and rapid—42 per minute, the pulse is weak and rapid—160 per minute The child's bowels move during the examination There is considerable straining and evidence of pain The stoolage consists of blood

and mucus—it contains no apparent fecal matter—the odor is not offensive. The mother says the child's bowels have been loose for several weeks, but that only within the last week have they contained blood The child has had fever for about one week For the last week the bowels move fifteen or twenty times a day The child has vomited but once during his illness The diagnosis is pellagra complicated by an acute ileocolitis

An examination of the other four children shows that each has a pellagrous eruption. The twin brother of the sickest child is chubby and fat, but he has a dirty looking pigmentation on the dorsa of his feet and on the extensor surfaces of his legs A sister, five years old, has an ulcerated sore mouth, bleeding gums, and a marked pigmentation on her feet and legs She also has a pigmented spot on either cheek. A sister, eight years old, has an erythema with a distinct line of demarcation on the dorsa of her feet, she has also a distinct Casal's collar The oldest girl, eleven years, has a sore tongue, red on its edges but pale and glazed elsewhere, she has an ulcer in each corner of her mouth, the backs of her hands are swollen and an erythema ends sharply 2 inches above the wrists, the eruption is also very distinctly marked on the dorsa of her feet. The father (examined June 22, 1917) has an eruption on the backs of his hands which extends 3 inches above the wrists The skin over these areas is of a bronze hue except where the skin is fissured, these fissures, about ¼ inch wide, are numerous, and in them the surface is raw and blood oozing The father does not use alcohol. He chews tobacco, but recently his mouth has burned and has been too sore to permit chewing without pain. His tongue is of the typical cardinal variety The mother chews snuff, but relates that for several days past she would as lief chew a live coal as to chew snuff Until the present summer no member of the family has been ill except that the mother has had "female trouble." The nature of this trouble is not ascertained, but is easily guessed when I am told that a country midwife presided at each of her confinements Prior to the present summer no member of the family has had an eruption or a sunburn severe

enough to attract attention No member of the family until the present season has had an intractable diarrhea.

It was regarded then that each member of this family was having his or her initial attack of pellagra For this reason and for the reason that not a single member in the family escaped, it was thought that these cases deserved a more or less intensive epidemiologic study

The Thompson-McFadden Pellagra Commission holds tena ciously to the opinion that pellagra is due to an infection, which it grants is probably low grade and not highly com municable, but is none the less an infection Members of the Commission admit that deficient dietary is an important pre-disposing factor, but they insist that this factor alone cannot explain the etiology of the disease The Commission has been impressed by the fact that new cases of pellagra develop either in the same house with or in close proximity to a pre-existing case, by the fact that endemic foci discovered during its investi gations were, as a rule, centered around localities where open privies existed or where there was careless disposal of human wastes, and by the fact that in whatever of these localities there was later installed a sanitary system of sewage at once occurred a marked reduction in the number of new cases of pellagra developing

The neighborhood in which the Johnson family lived was a poor one The house was a two-roomed cottage in an unsanitary alley Both rooms in the house were large and abundantly sup-plied with sunlight and air A roofed porch ran the length of the northern side of the house The house was unscreened The alley on which the house faced was about 40 feet wide and ran south from First Avenue to the railroad tracks, a distance of 300 feet The Johnson house was about 50 feet from the rail-road tracks, the space between, as well as a considerable area in front of the house, was open except for weeds Two other cottages of similar construction to the Johnson house were on the same side of the alley The shortest distance between any of these cottages was 15 feet The lots on which these cottages were built were not fenced in A four-room cottage facing

First Avenue on a fenced in lot 125 feet deep took up the remaining space on this side of the alley On the opposite, the western side, of the alley were two three-room cottages facing the alley, on fenced-in lots each about 25 x 50 feet, and a double three-room cottage facing First Avenue on a fenced in lot 125 feet deep The two cottages adjoining the Johnson cottage were occupied by negro families Five negro children and three negro adults were in the two families Across the alley one cottage was vacant and one was occupied by a very old gentleman and his young daughter No chicken, pig, or cow was kept on any of these premises A Belgian hare was kept by the Johnson children. Two dogs and a cat were kept by the negroes There was no crowding, but, on the other hand, plenty of air space for all the inhabitants of the alley The inside of the negro cottages was clean and neatly kept. Both were unscreened The negroes were careless in the disposal of waste waters and table refuse There was no well in the neighborhood, each cottage being supplied with city water

To the rear of each cottage was what was probably originally a sanitary privy The construction of the privies was practically the same. It consisted of a wooden frame with one and in some instances two seat holes Under each hole and standing in a wooden box was a tin or galvanized iron receptacle. The rear of each privy box had a hinged frame flap or drop intended to be kept down except at the time of removal and emptying of the receptacle in order to prevent flies, pigs, chickens etc., from having access to its contents The hinges on the flaps on nearly all the privies were found intact, but in several instances they were found only part way down Covers for a number of the seat holes in the privies inspected were found detached, and in a number of other instances they were not down in place Many of the receptacles were found exposed and in a bad condition This kind of a privy is attached to not only the cottages in the alley, but to all the cottages facing First Avenue for a distance of one block on either side of the alley Their number was not counted, but was estimated to be between twenty five and thirty Eight of them were inspected by me, with findings as mentioned

above The receptacles were emptied once a week The capacity of the receptacle was about 15 gallons Some of them were running over at the time of inspection The privy on the Johnson lot was found to be in a worse condition than any The rear flap was prevented from closing by a large stone intentionally or accidentally placed under one end The receptacles were brimful and had slopped over The seat-covers were detached and missing The door was on one hinge The privy inside and out was swarming with flies Tall weeds surrounding the Johnson cottage offered conveniences which were sometimes at least preferred by the children in the family to the accommodations of the privy To this fact my shoe at the time of my first visit bore convincing olfactory as well as ocular evidence

So far my findings fitted well into the ideas of the Thompson McFadden Commission with reference to the spread of pellagra When I discovered 3 other cases of the disease, each of which gave a history of having the initial attack in the summer of 1916, living in the neighborhood, I was at first quite sure that the communicability of pellagra was going to be proved at least to the satisfaction of my own mind These 3 cases were the McGrady children, two girls and a boy The McGradys lived in one side of the double cottage facing First Avenue, the 125 feet lot of which formed the western side of the alley Figure 270 shows a diagram of the alley with its privy and cottage arrangement

Fuller investigations by me, however, weakened the evidence pointing to the communication of pellegra to the Johnsons Aside from the members of the Johnson family, each of whom was experiencing the initial attack, and the three McGrady children, each of whom was experiencing the second attack, no other case of pellagra existed within a radius of two blocks If the disease was communicated to the Johnsons, then it must have been communicated from the McGradys The McGrady privy was not less than 100 feet from the Johnson house The privy was on ground which drained away from the Johnson house Of all the privies inspected, the McGrady one was in a sanitary condition The rear flap was kept tightly down The receptacle

was in good condition, was without leaks, and was carefully placed. Lime had been used inside and around the receptacle

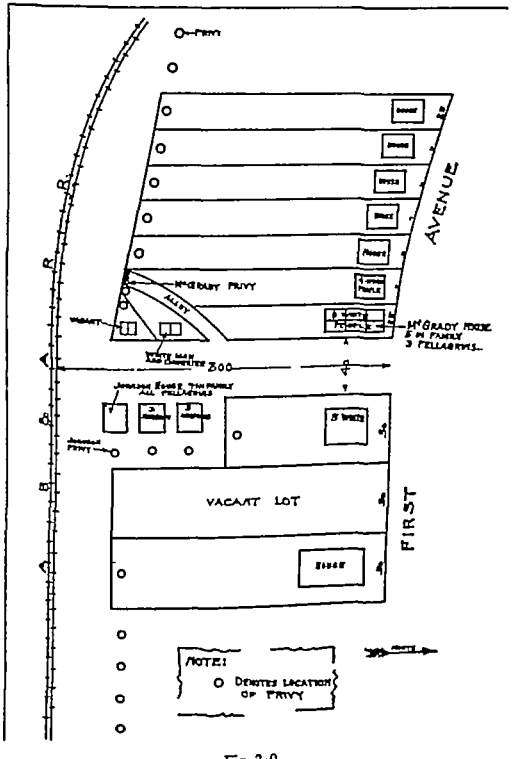

Fig 2.0

The seat-cover was down The door was kept shut. The McGrady father was a motorman His wife was intelligent and

was evidently a good manager The house was screened and as clean and neat as a new pin Father and mother and the three children constituted the household The McGrady children were clean and neatly dressed Although the McGrady children sometimes played with the Johnson children, there was no great intimacy, as the McGrady mother tried to discourage this Without the offices of some insect, as a carrier, the McGrady privy did not impress me as being a probable factor in the spread of the disease to the Johnsons There were fewer bugs and insects on the McGrady premises than anywhere else in the neighborhood A few flies were found on the outside of the privy One was seen on the inside The scarcity of flies inside the closet was due to its darkness when the door was closed In the alley itself flies of the ordinary domestic variety were numerous Unidentified mosquitos, grasshoppers, and small yellow butterflies were also seen Insects and bugs as a possible etiologic factor in pellagra were studied by Jennings and King for the Thompson-McFadden Commission By convincing methods, these gentlemen were enabled to eliminate every one of the many insects which have from time to time been suggested as being in any way connected with the etiology of pellagra For a time Jennings and King believed that the stable-fly might have some relationship to the disease Repeated experiments conducted by them failed to sustain this opinion

Similar though some of my findings relative to pellagra in the Johnson family were with the findings of the Thompson-McFadden Commission, I was unable to arrive at conclusions similar to those held by members on the latter body

On the other hand, the investigations relative to the diet of the Johnson family and of the three McGrady children seemed to point very strongly to the correctness of Goldberger's conclusions Goldberger, who believes that pellagra is in no way communicable, is of the opinion that the disease is the result of some dietary deficiency Those of you who are unfamiliar with or rusty on the subject of dietary deficiencies are referred to the writings of Funk, Voegtlin, Vedder, etc. These men believe that in the well-balanced diet of man there are, in addition to the

commonly recognized essential elements, a certain number of specific disease-protecting elements—the so-called accessory factors or vitamins. When man lives on a diet from which any one of these vitamins is absent he will develop either beriberi or scurvy, or rickets or pellagra, depending on whichever one of the vitamins is absent. It has been definitely established that beriberi and the less commonly known disease, xerophthalmia, is each the result of living on a diet from which is absent its specific vitamin. These, then are true deficiency diseases. So far theory alone classifies scurvy, rickets, and pellagra along with them. McCollum and his co-workers, who dispute the existence of a pellagra-protecting element or vitamin and the existence of a scurvy-protecting element or vitamin, believe that pellagra is the result of several factors, chief among which are mechanical injury to the mucosa of the intestine from continued use of an appropriate food-stuff and the resulting stimulus to bacteria ordinarily not numerous in the intestines.

There will be no attempt here to reconcile the scientific points. As stated, the study of the diet of the Johnsons and of the McGrady children as made by me pointed to the correctness of Goldberger's conclusions. But I referred only to Goldberger's general and broader conclusions, namely, that pellagra is not communicable, but is the result of the continued use of a diet lacking in animal and vegetable proteins.

The Johnsons, as you will recall from the history, had lived in the country until September, 1916. While in the country they had no near neighbors. While in the country each member of the household had remained well. While in the country the family kept a cow and raised chickens. The mother told me that while in the country milk and eggs formed a part of almost every meal. Fried or boiled or baked chicken was served from time to time. Occasionally fresh pork was served. Occasionally fresh goat meat was served. So that though the chief meat even while in the country, was salt pork, fresh meat was not unknown to their stomachs. The table fare after removal to the city has already been discussed. But the significance of the contrast between the food of the family while in the country and

after moving to town cannot be appreciated fully without a consideration of the findings relative to the diet of the McGrady children

The McGradys were in no such financial distress as were the Johnsons The McGrady income was sufficient to provide something more than the bare necessities My offer to have milk furnished to the McGrady children out of a fund provided for by one of the daily newspapers of the city was declined Mrs McGrady told me that the husband and father was able and willing to amply provide food for his children Investigation, in fact, showed that such provision was made Investigation also revealed the fact that of the three McGrady children not one liked meat, milk, or eggs The chief articles of diet with these children, a boy five years old, and two girls, seven and ten years of age respectively, were cereals, fruits, bread and syrup, battercakes and syrup, and green vegetables cooked with fat meat. In order to induce these children to drink milk and to eat eggs and meat it was necessary to offer rewards and prizes It was only after several weeks of reluctant acceptance of these latter articles that a taste for them was cultivated Therefore, though the sanitary conditions in this neighborhood were bad, though the conditions of the privies should be regarded as intolerable, though pre-existing cases of pellagra were living fairly near to the new cases, it seems to me that there was little relationship between the new and the old cases except that in both was discovered a diet deficient not in quantity but deficient in protein content.

DISCUSSION

THE PRESIDENT Will Dr Snyder tell us the outcome of these cases?

DR SNYDER (continuing) Every one of the cases referred to by me in the report gained weight, had symptoms disappear, and made an apparently complete recovery in a very few weeks Except for a dose of castor oil, administered to the sickest child, no medicine was used The treatment was entirely dietetic and consisted in incorporating in the diet of each a larger percentage of proteins, as furnished by milk, fresh meat, and eggs

Except in the case of the sickest child nothing was said about what should not be eaten Corn bread, cereals, white meat, syrup, batter cakes, etc., of which they had previously partaken, were not prohibited In fact, I remember saying to the McGrady mother that if pellagra was of dietetic origin it was caused not by what was eaten, but was caused by what was not eaten. The Johnson father, on account of his increased capacity for work, was given a promotion with an increased salary Toward the end of the summer the family moved to another community Up to the time of their departure they were all well. I have heard indirectly that they continue in good health The McGrady children continue under observation. So far there is no evidence of a recurrence in any one of them

Dr. D L Wilkinson Mr President, I do not know that I endorse in its entirety Dr Goldberger's conclusions as to the cause of pellagra or his dietary treatment. I have seen and treated a good many cases I have known several fairly well to-do farmers who largely lived on milk, eggs, and chicken, and who still went on and died from pellagra. I remember in particular two brothers who were in this class, both large, healthy men, except for living in a malarial section and giving a previous history of malaria, who while they lived largely on these products and what they grew at home, still they developed pellagra and died from it in spite of Goldberger's dietary In my experience I have seen so many cases secondary to some other trouble that at one time I almost concluded that pellagra was always secondary to some other trouble. I have perhaps treated 25 cases of pellagra and had a large percentage of them to go on and die in spite of either medicinal or dietetic treatment. I wish to thank Dr Snyder personally for his very able and thorough paper

Dr W B Hardy Hearing Dr Snyder discuss the diet in pellagra reminds me of a four year-old boy we had in the hospital recently with a second degree burn of the right side of his body

In due time the surface was partially covered with skin grafts, as we were unable to secure sufficient skin to cover it entirely, but, for some reason, the skin did not thrive and take hold as we had hoped it would Two weeks later the grafts

seemed perfectly healthy, but showed no signs of spreading At this time he was seen by Dr Snyder, who pointed out to us definite signs of pellagra on his knees and wrists At Dr Snyder's suggestion in addition to his usual diet he was given milk and eggs in abundance

After a few days it was indeed wonderful to see the change in the general condition of the child, and especially the grafts, which began to spread, and a few weeks later he was able to leave the hospital, the entire surface having healed over without further grafting

DR H S WARD I enjoyed Dr Snyder's case reports and congratulate him upon their thoroughness They are very instructive, and to me are further proof, if it were needed, that pellagra is entirely a disease of improper diet. An exclusive diet of carbohydrates, green vegetables, and "white" or fat meat will produce pellagra I see a great many cases of pellagra, and if one will take a thorough history and get the real truth of what the patient actually eats—not what was on the table—each can be traced to a "poor folks" country diet of carbohydrates, green vegetables, and fat meat

I recently saw a real estate man who said that his rental income was $700 per month, and that his family had a liberal diet, but when pressed as to what he really ate, it consisted of cereals, coffee, syrup, and cakes for breakfast, usually rice pudding or some form of sweets for lunch, bread, butter, syrup and preserves, and coffee for supper He said that milk, eggs, or meats gave him indigestion He had definite and well-advanced pellagra—diarrhea, sore mouth, etc. This man had been given sodium cacodylate for months, with the assurance of a certain cure His diarrhea and other symptoms were getting progressively worse Diet had not been mentioned to him A diet of milk and raw eggs only soon made a well man of him

A mother with two small children, all three with pellagra, gave a history of having been far from a market and had no cow or chickens, so were compelled to live on green vegetables, bacon, and carbohydrates She and children went to her mother's farm and ate milk and eggs only for three weeks, and returned

to my office Every apparent symptom of pellagra had vanished. In fact, I did not recognize them at first. These were early acute cases, and responded more quickly to diet than the chronic ones.

Any diet with proteins in excess will perhaps relieve pellagra, but they are all fond of carbohydrates, and it is so easy to take more than is good for them. The only medicine I give in the ordinary cases is dilute nitric acid, if diarrhea is severe, otherwise dilute hydrochloric acid Observation has shown that pellagrins secrete very little hydrochloric acid, so I feel that this is only supplying a physiologic need. Arsenic is of no value, *per se*, and should be tabooed Whether the diet theory is correct or not, if it cures, let's give our patients the benefit of it until some one is able to *prove* something that is better

DR W G HARRISON Recently while attending a meeting of Medical Directors of One Hundred Life Insurance Companies the question of goiter in its relationship to life insurance was up for discussion, and one of the physicians present who was from eastern North Carolina made some very interesting remarks He said "the death rate from his company from pellagra was higher than from any other one disease, and that the death rate in this section of the country from pellagra was definitely higher than from tuberculosis or pneumonia." He spoke of having seen a great many cases of pellagra associated with goiter, and expressed himself as believing that there was some association between the two diseases He stated frankly that he had no scientific data on which to base his suspicion, but having seen these two diseases associated in so many cases he was asking other men to watch carefully for such connection, and to report if they found similar relationship I have forgotten his exact statement, but I think he said that he had noticed an association of the diseases in several hundred, maybe more than a thousand, cases

DR. SNYDER (closing) This report was presented without ambition on my part to settle the etiology of pellagra. Pellagra is a most fascinating study which any one who stops guessing at the etiology will admit If each of us will investigate every case of the disease that is met with, the accumulating mass of data will sooner or later result in a definite settlement of etiologic

points If my report will stimulate study among us, I will feel that it has been worth while

With reference to pellagra being a secondary disease, I want to say this In malarious districts malaria and pellagra are frequently concomitant diseases, in districts where uncinariasis is prevalent hookworm and pellagra are frequently concomitant, in districts where amebiasis is common pellagra and amebæ are frequently linked together, in some districts corn whisky is the concomitant disease, and tonight we have heard from Dr Harrison that in some localities goiter and pellagra are concomitant Now if we happen to be living in one of these districts and do not look over to see what is occurring in the other districts, we are apt to reach the conclusion that pellagra *is* a secondary disease, we are apt to believe that the particular prevailing infection in our locality has a definite etiologic connection with pellagra But if we broaden our outlook, I think it becomes perfectly plain that neither malaria, nor amebæ, nor hookworm, nor corn whisky, nor goiter has any more definite connection with pellagra than any other enervating and devitalizing agency

CLINIC OF DR J HEYWARD GIBBES

COLUMBIA HOSPITAL COLUMBIA S C

HODGKIN'S DISEASE

Primary Involvement of Mediastinal Glands with Presentation of Tumor Mass Over Sternum Blood-picture Characteristic of Well-advanced Disease Differential Diagnosis Etiology Pathologic Anatomy Prognosis. Treatment.

THIS man is thirty six years old a farmer by occupation He first came under my observation one week ago complaining of "swelling on chest loss of weight weakness and cough

His family and marital history is unimportant

In his personal history he says that he had measles, whooping cough, and malaria in early life and that up to two years ago he drank excessively He denies all venereal infections says that he has not been troubled with sore throats and gives no indications of previous tuberculous infection He maintains that up to the onset of his present illness his health had been unusually good

He dates his present illness from twenty months back when he was taken suddenly with a stitch in the left side of chest which was intensified by deep breathing This pain gradually disappeared, but he soon developed a mild but persistent cough without night sweats or fever He very shortly found that he was losing strength and weight. About five months ago his attention was attracted by a swelling over the sternum extending over toward the left breast, and which he describes as soft and resembling an abscess" He consulted a physician who inserted a needle into the mass, but failed to obtain fluid A few days later an incision into the mass was made and a material expressed

which "looked like brain matter" Since that time he has progressively lost weight, has become increasingly weaker, and has been troubled with a persistent and productive cough At no time has there been blood in his sputum The cough is distinctly worse at night and when patient is in prone position

Upon physical examination we see a man of large frame, evidently emaciated, and with marked pallor of skin and mucous

Fig 271

membranes There is considerable hypertrichosis over chest and forearms A few purpuric spots are visible over deltoid regions and chest This large tumor mass presenting just to the left of the midsternum measures approximately 20 cm in transverse diameter and 4 5 cm in the other dimension The superficial ulcerated area on the apex of the tumor represents the site of

certainly no excess of fluid in the flanks The lower extremities show nothing of note, but the fingers present a beautiful example of clubbing and watch-crystal nails, the condition classically known as Hippocratic fingers (Fig 272), and more recently described as a chronic pulmonary osteo-arthropathy

The sputum is very tenacious, mucopurulent, and abundant, being raised especially in large quantities in the early mornings Careful studies for tubercle bacilli have been negative No microscopic or macroscopic blood has been observed

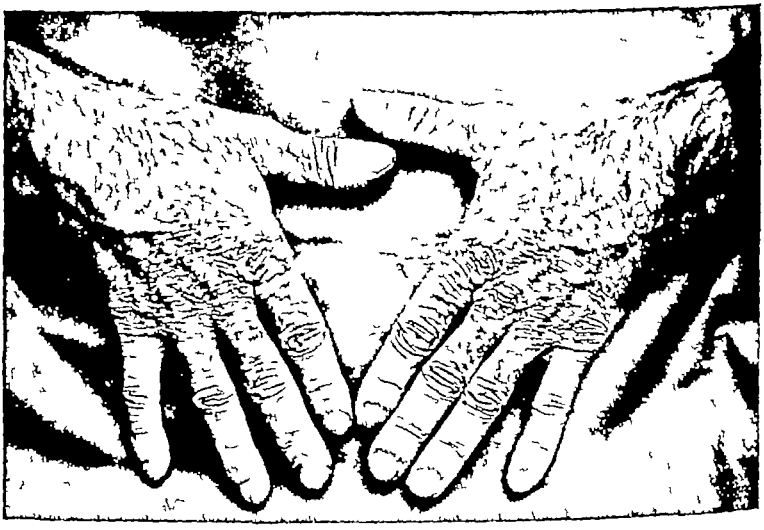

Fig 272 —Hippocratic fingers

The urine is of some interest in that it contains an abundant, granular sediment which disappears on heating and reappears upon cooling—a heavy deposit of urates These amorphous salts are visible under the microscope, and upon the addition of hydrochloric acid large numbers of uric acid whetstone crystals make their appearance It is interesting to think that this finding in the urine is probably dependent upon the chronic leukocytosis present in this case, there being a constant destruction of the nuclei of large numbers of cells Careful heating of the urine in a water-bath failed to disclose the presence of the

Bence-Jones protein As you doubtless know, this body was formerly supposed to be practically pathognomonic of the disease known as multiple myelomata, a multiple tumor occurring in the bones But recent studies, notably those of Boggs and Guthrie, from the Johns Hopkins Hospital, have shown that the Bence-Jones body is present in the urine in carcinomatosis of the bones, myelogenous leukemia, and less constantly in cases of lymphatic leukemia While the condition which we have represented by this patient is not primarily a disease of the leukopoietic tissues, there is at present, as you will see from the report of the blood studies, a functional involvement of these structures, and it would not have been surprising to find the Bence-Jones body in this urine

The blood of this patient is of great interest because of the variety of cells which it presents an opportunity to observe and because of the statement of Bunting that we find here specific criteria for the making of a diagnosis. You will find a stained preparation under the microscope which I hope you will all look over at the end of the demonstration The following is a summary of the blood findings

I. The fresh blood
   1. No parasites.
   2. No poikilocytosis or anisocytosis.
   3. Red cells much paler than normal.
   4. There is an evident leukocytosis with many coarsely granular cells.

II. Red blood-cells, 3,800,000.

III. White blood-cells 26,000.

IV. Hemoglobin, 65 per cent. (Sahli)

V. Differential count

1	Polymorphonuclear neutrophils	925	92.5 per cent.
2	Polymorphonuclear eosinophils	15	1.5 per cent.
3	Polymorphonuclear basophils	00	0.0 per cent.
4.	Large mononuclears	19	1.9 per cent.
5	Lymphocytes	23	2.3 per cent.
6	Transitionals	15	1.5 per cent.
7	Myelocytes (neutrophilic)	3	0.3 per cent.
		1000	100.0

In counting these cells 2 nucleated red blood-cells were observed.

We have here a marked secondary anemia, a pronounced leukocytosis, a remarkable relative increase in the polymorpho-

nuclear elements, and a slight relative increase in the eosinophils Another point of interest and importance, which you may observe in the preparation under the microscope, is the large number of platelets present and especially the unusual size of many of them Bunting is of the opinion that these large platelets probably are of recent formation through the breaking off of the pseudopodia of the megalokaryocytes in the bone-marrow, and they constitute an important diagnostic link in the chain of evidence to be derived from the study of the blood

The studies of Bunting and Yates concerning the blood-pictures of Hodgkin's disease are the most interesting observations in connection with this subject since the work of Dorothy Reed put the disease on a sound basis of pathologic anatomy Among the most interesting papers by Bunting are those published in the Johns Hopkins Hospital Bulletin in 1911, pages 114 and 369, and in the Journal of the American Medical Association in 1915, page 1953 The above blood-picture agrees in detail with that described by Bunting as occurring in the late stages of Hodgkin's disease The leukocytosis has been found in some instances to reach as high as 100,000

The Wassermann reaction is negative

The conjunctival tuberculin tests, 1 and 5 per cent., are negative

The *x*-ray picture of the chest shows here a dense, broad mediastinal shadow, a marked clouding of the left lower lobe, a more dense infiltration at the level of the upper border of the left scapular, and numerous, well-defined, discrete nodules scattered throughout both lungs, suggesting an involvement of the bronchial lymphatics

We will now allow the patient to retire

The condition which we have just seen must be differentiated between

1 Syphilis
2 Tuberculosis
3 Lymphosarcoma
4 Hodgkin's disease

The Wassermann, tuberculin reactions, sputum studies, and the

blood studies virtually dispose of the first two conditions In neither syphilis nor tuberculosis would one expect to find the leukocytosis or the high percentage of polymorphonuclear cells which is present in this case, even in the presence of a secondary pyogenic infection It would be most unusual to say the least. As regards the possibility of differentiation between lymphosarcoma and Hodgkin's disease in this patient, one must confess to an element of doubt on the basis of clinical evidence alone. We certainly should not be satisfied without the histologic examination of the diseased tissue. The patient has thus far refused to submit to such examinations Had the patient come under observation in the early stages of his disease, before the leukocytosis had appeared and when the differential blood count may have shown a relative increase in the mononuclear elements, we should probably have had to consider a fifth condition, that of lymphatic leukemia. It is seldom, however, that the blood of Hodgkin's disease approaches the relative mononucleosis seen in lymphatic leukemia Accepting Bunting's standards of blood interpretation, we may safely conclude that we are dealing here with a truly remarkable example of Hodgkin's disease, the primary involvement having taken place in the glands of the mediastinum.

The etiologic factors in this disease are relatively unknown Renewed interest has been added to this phase of the subject by the work of Bunting He has succeeded in recovering from the glands and adjacent tissues a non acid fast, Gram-positive, diphtheroid organism which he believes to be the causative agent of the disease. He reports the production of a glandular disease in monkeys through the agency of this bacterium, and believes that he has obtained good clinical results from the use of the organism as a vaccine. However, one must await further developments in this connection before finally accepting these views. Pleomorphic, diphtheroid organisms, very similar to those described by Bunting and his co-workers, can frequently be obtained from lymph glands in other conditions than Hodgkin's disease. In this connection it is well to keep in mind a similar piece of work done with reference to pellagra here the isolation

of the diphtheroid organism from the glands of patients, its injection into monkeys, and the reported production of a pellagroid condition in these animals It is to be hoped that Bunting's observations are on a firm foundation, for with their substantiation will come increased information and the possibility of an effective therapy

The treatment of Hodgkin's disease is essentially empirical. The disease is characterized frequently by remissions in which the patient seems to be substantially improved, and it is probable that this may occur independently of therapy Arsenic seems at times to have a beneficial effect, especially in the form of salvarsan, and it is well to give a few injections of this drug x-Ray treatments over the affected glands should always be practised, needless to say, with discretion Sustaining measures, good food, proper hygiene, iron in some form in the presence of anemia, and the treatment of symptoms as they arise are practically the only measures at our disposal The removal of tonsils as possible portals of entry for the exciting agent of the disease and dissection of the affected glands in the early cases have been advocated and may be tried under propitious conditions

REFLEX GASTROSPASM

Differential Diagnosis Between Functional Spasm and Organic Deformity of Stomach

THE patient that I wish to use as a text for the clinic today is sixty two years of age. She was referred to me for study by Dr Guerry, having come to him with the tentative diagnosis of ulcer of the stomach, with probable early malignant change.

I saw her first in August, 1917, at which time she stated that she had been gradually losing strength for about one year She said that she had been having some little trouble with her stomach for the past ten years, gas, heartburn, and slight pain after eating For the past four months the pain had been particularly bad, coming on immediately after eating, and the gas had been most annoying There had been no nausea or vomiting, no hematemesis, and no melena She had lost about 20 pounds during this period In addition to this, she complained of pain and burning in the "neck of her bladder"

Her *family history* was unimportant, with the exception of the fact that she had given birth to one still born child

The positive points of her *personal* history were Typhoid fever at twelve years of age, indefinite pains in the joints at times, headaches and dizzy spells occasionally, and the loss of all of her teeth as the result of pyorrhea

On **physical examination** she was found to have a poor color, and presented evidences of a considerable loss of weight There was a slight yellowish cast to the skin, but no distinct jaundice The eyes were not unduly prominent, the pupils were equal and reacted normally There was a well marked pterygium on both eyes, more developed on the left. All of the teeth had been removed The throat was negative. The heart and lungs presented no abnormalities The abdomen looked natural, no visible masses or peristalsis The walls of

the abdomen were fairly soft except for slight muscular resistance on palpation over the epigastrium, the patient being distinctly "sore" over the entire epigastric area This sensitiveness seemed somewhat more marked under the right costal margin No masses were felt in the abdomen, but an indefinite sensation of a palpable gall-bladder was elicited The spleen and liver were not felt, and the stomach seemed to be in a high position on auscultatory percussion The extremities were negative Vaginal examination disclosed a cauliflower hemangioma just outside of the urethral orifice. The uterus was of normal size and in good position Her blood-pressure was 160 systolic and 80 diastolic, the pulse 88, and her temperature reached 99° to 99½° F each day

The urine showed a specific gravity of 1020, a trace of albumin, no sugar, and a good many pus-cells under the microscope. A catheter specimen could not be obtained because of the obstruction caused by the caruncle

The stool gave a positive test for occult blood on one occasion, but was negative on other examinations

Her blood showed 12,000 leukocytes and a hemoglobin of 75 per cent. by a Sahli instrument

In the gastric analysis we recovered 60 c.c of an Ewald test-breakfast at the end of one hour The free hydrochloric acid content was 28 and the total acidity 45 There was no lactic acid present, no blood, and the microscopic examination was negative

The Wassermann reaction was negative

The bismuth pictures of the stomach showed a "cow-horn" stomach with incomplete filling of the pylorus and duodenal cap There was rapid expulsion of the bismuth into the small intestine The filling defect in the pyloric region was persistent in three pictures

On the basis of these findings, we felt that we were dealing with a chronic cholecystitis which was giving rise to reflex gastric manifestations, and advised exploration of the gall-bladder and pyloric regions Operation was performed by Dr Guerry on the 29th of August, and a well-defined cholecys-

tus with a stone in the cystic duct was found The stomach presented no signs of pathology The gall bladder was removed With the exception of an acute pyelitis developing the third day after operation, the patient had an uneventful postoperative course

The patient returned to the hospital on March 25, 1918, for the purpose of having the urethral caruncle removed She says that she has gained weight since her operation and has been practically free of epigastric pain She has, however, continued to have a considerable amount of "gas on the stomach" and mild nausea at times She thinks that she has had some fever each day since leaving the hospital, reaching as high as 101° F at times

The physical examination at present differs in no way from that of last summer except for evidences of increase in body weight, the absence of tenderness in the epigastrium, and a palpable right kidney The gastric analysis shows essentially the same findings, and the persistent low fever is explained by the findings in the urine indicative of a chronic pyelitis

The *x* ray picture of the stomach at present is of particular interest, showing in every respect a normal organ, as contrasted with the persistent pyloric deformity which was manifested prior to the gall-bladder operation.

The urethral caruncle was removed by Dr Guerry on the 2d of this month

Here we have an illustration of that interesting group of cases in which the presenting symptoms are gastric, and in which these symptoms may be of such a pronounced character as to completely obscure the primary pathologic condition In this patient the diagnosis of gastric ulcer had already been made and the fear of gastric cancer expressed The fact that we were able to arrive at an accurate determination of the cause of the gastric phenomena observed in this case does not imply that we are equally as successful in all others, or, in fact, that we generally succeed in differentiating organic disease and functional disorders of the stomach In collaboration with Dr R W Gibbes and Dr Guerry, it has been my good fortune to investigate a large

number of patients with presenting gastric symptoms during the past few years, and we have found here a field of unlimited interest and never-ending surprise In many respects the roentgenographic and roentgenoscopic study of the stomach by means of the bismuth or barium meal is the most exact means of investigating this organ But this method furnishes us with some of the most confusing data with which we have to contend This is borne out by the series of pictures that I have just shown you

These pictures exhibit an excellent example of pylorospasm, with some extension of the spastic condition into the prepyloric portion of the stomach The frequency of involvement of the distal third of the fundus in these spastic states has suggested the term "gastrospasm," as distinguished from the term "pylorospasm" when the spastic condition is limited to the pylorus The roentgenologists were not slow in recognizing these so-called "reflex" spasms of the gastric musculature, and an extensive literature has accumulated on the subject One of the most illuminating articles is by Cole, in the American Journal of Medical Sciences for July, 1914 Practically every one is agreed that it is by no means a simple matter to distinguish at all times between the deformity of the stomach produced by spasm and that resulting from ulcer or tumor encroachment upon the stomach wall In probably the majority of instances the spastic deformities are inconstant, and serial pictures or fluoroscopic observation will reveal complete relaxation or at least some change in the type or degree of the deformity But we have now seen a large number of reflex gastric spasms which were so persistent as to defy our efforts at differentiation by means of *x*-ray study The deformities resulting from ulcer and malignant disease are, of course, relatively much more constant But gastric spasm frequently occurs in association with organic disease of the stomach, and we have seen this state of affairs so well marked as to render accurate interpretation practically impossible A point of interest in this connection is the fact that prolonged reflex spasm of the stomach muscle will result in hypertrophy and induration of the wall to such an extent that a permanent deformity will be evidenced in the *x*-ray plates

Fig 273.—Picture of normal stomach with well formed pylorus and complete filling of duodenal bulb, no filling defects.

Fig 274.—x Ray picture of stomach showing spastic condition of pylorus with marked filling defect. Patient with chronic cholecystitis and stone in cystic duct. No pathology at operation

Fig 275 —Showing persistence of pylorospasm.

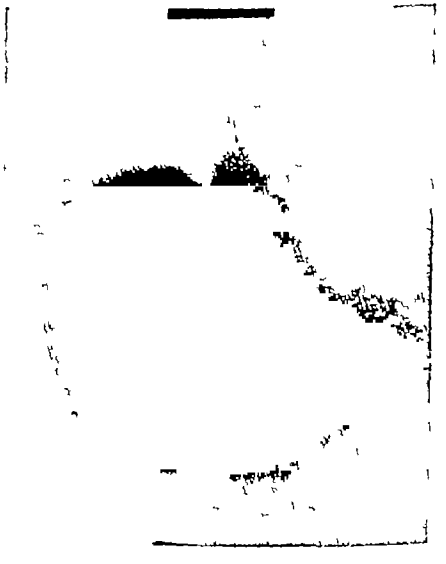

Fig 276 —Picture of same stomach eight months after removal of gall bladder, showing normal filling of pylorus and cap

Fig 277.—Spastic pylorus in presence of [illegible] appendicitis and cecal stasis

Fig 278.—Cecal region and transverse colon with bismuth in appendi

The symptomatology of reflex gastrospasm or, more properly, of the functional disorder of the stomach which is associated with the gastrospasm, is essentially that of ulcer I have seen these patients complain of pain immediately after eating, gas, acid eructations, and epigastric hyperesthesia Others will give a typical story of duodenal ulcer The symptomatology alone is often of little help But too much attention cannot be paid to the taking of a systematic and thorough history, enquiring carefully into the present or past incidence of conditions that might give rise to functional disorders of the stomach These conditions are, briefly chronic infections in the respiratory tract, oral sepsis, cholecystitis or cholelithiasis, chronic appendicitis, pelvic inflammatory disease, or peritoneal adhesions Psychoneurotic states may add confusion to the picture

The physical examination does not always serve to lighten our difficulties As in the present case, we may be so fortunate as to have it point more or less clearly to a chronic infection in the gall-bladder or appendix region, but too frequently we find the patient with a punctate point of tenderness over the pylorus with a radiation of the pain into the back and a hyperesthesia over the epigastrium At times the pylorus may be distinctly felt and give the impression of an epigastric mass

In the majority of these cases the gastric analyses have shown a degree of acidity on the upper limit of normal, a few of them have shown a very high free hydrochloric acid content, and one patient exhibited an absence of free acid In none of them has blood been found in the gastric contents, but do not lose sight of the fact that slight trauma from the tube might introduce this confusing element The fractional estimation of stomach contents—*i e*, the removal of portions of a test-meal every fifteen minutes after its introduction until the stomach is empty—is putting our knowledge of normal and abnormal gastric function on a more exact basis, and may in time furnish an answer to some of these puzzling questions But I need only call your attention to the statement of Crohn and Reiss in an article in the American Journal of Medical Sciences for December, 1917, to the effect that they have been unable to differentiate the

curves of gastric acidity seen in cholelithiasis and chronic appendicitis from those found in ulcer of the stomach.

Repeated examinations of the [illegible] for occult blood is of decided help in differential diagnosis. Repeatedly negative tests for occult blood are most valuable in attempting to eliminate gastric ulcer or cancer from consideration. But bear in mind that positive tests are of much significance. The diet should be practically hemoglobin and the nose, pharynx buccal mucous membrane and [illegible] investigated as possible sources of blood before a positive is allowed to carry its full weight.

In my experience the most helpful single examination has been a leukocyte count. The cases of reflex gastric spasm associated with chronic cholecystitis, chronic appendicitis or chronic pelvic inflammatory disease have usually shown a mild leukocytosis. But here again one must keep in mind the possibilities of error. A gastric ulcer is frequently the seat of secondary infection and a high leukocytosis is often associated with malignant disease.

Gastroscopy has not come into general clinical use as rapidly as one might have expected, doubtless because of the technical difficulties incident to its application. In the opinion of such men as Janeway, of New York, and Jackson, of Pittsburgh, this procedure furnishes very exact information concerning the cardiac end of the stomach and they think that it takes precedence over all other means of investigating this portion of the organ, just as the x-ray does for the pylorus. Not infrequently it is necessary to give an anesthetic for the introduction of the gastroscope, but in careful and experienced hands the method can be carried out with perfect safety. While reflex spasm of the cardia is much less frequently encountered than pylorospasm it does occur at times and direct vision should enable us to eliminate organic disease from consideration.

I wish to call your attention briefly to another group of cases, the exact pathology of which is very uncertain, which present manifestations readily confused with ulcer or which might frequently be interpreted as a reflex gastrospasm. During the past eighteen months I have seen 4 patients with typical

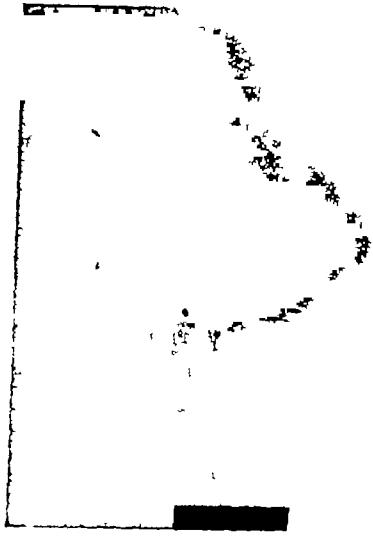

Fig 279.—Patient eighteen years of age, history suggestive of gastric ulcer with four plus Wassermann reaction Condition thought to be ulcer until report on Wassermann was obtained

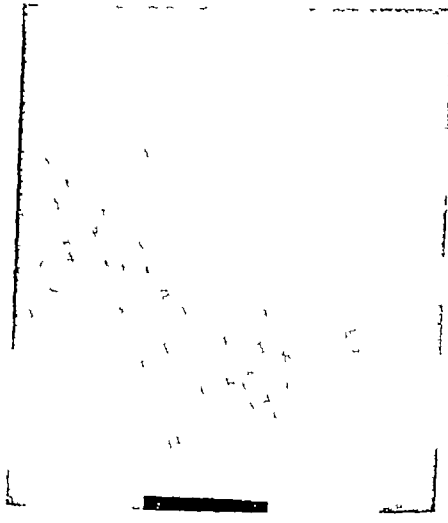

Fig 280.—Showing persistent pyloric defect in above case

ulcer histories, 2 of them having hyperacidity findings in the gastric secretions, one a hypo-acidity, while in the fourth no gastric analysis was obtained All of these patients gave positive Wassermann reactions, and their stomach symptoms promptly disappeared under salvarsan and mixed treatment. You will note in one of the pictures here a persistent filling defect in the fundic portion of the stomach, while the other case shows a deformity in the pyloric region which was regarded as a reflex spasm until the Wassermann report was obtained Unfortunately, we have been unable to get pictures of these stomachs since the administration of treatment We hope to do so in the near future, and it will be interesting to see what changes have taken place It is quite possible that these cases represent an actual syphilitic ulceration of the gastric mucosa, either as mucous patches or as ulcerative degeneration of gummata Proliferative infiltration of the gastric wall and cicatricial deformity of the lumen have been observed at operation on patients with syphilis of the stomach The routine application of the Wassermann reaction is leading to the discovery of more and more gastric syphilis Take the temperature and take the pulse, but also take the blood for a Wassermann reaction, is advice that we all might follow with advantage to ourselves and to our patients

I hope that I have shown you some of the difficulties of gastric diagnosis In possibly the majority of cases it is possible to differentiate between functional and organic conditions, but a considerable number of cases will defy this distinction Given this primary decision, say, for example, that we recognize an organic deformity of the stomach in a certain case, the differentiation between benign ulcer and malignant disease is a relatively simple matter But you will find, no matter how careful and exhaustive your investigations may be, that you will mistake functional spasm for organic deformity many times, and vice versa The greater number of reflex gastrospasms result from intraperitoneal pathology, especially chronic cholecystitis, chronic appendicitis, and pericecal adhesions, and all of these conditions are to be looked upon as surgical when

associated with profound derangements of gastric function. I think you might accept the following as a safe rule A history of gastric malfunction, gastric secretory findings deviating from the normal, an *x*-ray plate indicating anomalies of motor function in the stomach, and the elimination of extraperitoneal causes of gastrospasm, together with a negative Wassermann reaction, represents a group of clinical data indicating the necessity for exploration of the peritoneal cavity

CONTRIBUTION BY DR WILLIAM H. DEADERICK

VISITING PHYSICIAN, LEO N LEVI MEMORIAL HOSPITAL, HOT SPRINGS, ARKANSAS

THE TREATMENT OF MALARIA

IN the treatment of malaria we possess a drug which is probably the type of specifics, quinin Most of the salts of quinin are readily absorbed from the stomach It has been shown, however, that the tannate is more largely absorbed from the small intestine The rapidity of absorption varies with the different salts and is estimated by the length of time required to appear in the urine. This varies from fifteen minutes with the hydrochlorid to three hours with the tannate.

The method of administration of quinin influences the rapidity with which it is absorbed Given by mouth, a highly soluble salt will begin to appear in the urine within fifteen to thirty minutes, and is eliminated in the greatest quantity within three to twelve hours Employed hypodermically, the rapidity and thoroughness of absorption depend upon the solubility of the salt and the concentration of the solution. The latter is of the utmost importance, since no matter how soluble the salt, if given in concentrated solution it will not be absorbed After intravenous administration quinin may be detected in the urine in ten to twenty five minutes

The action of quinin upon the malaria parasites is that of a protoplasmic poison, and is most powerful against the spores before they have assumed the protection of the red blood-cells While young and half grown tertian and quartan gametes are sometimes destroyed by quinin, those of the estivo-autumnal variety are exceedingly difficult to kill In rare instances small doses of quinin appear to arouse latent malaria, a provocative

reaction, and in isolated cases there is no doubt that the parasites acquire a relative immunity to the drug and become quinin-fast.

The mere statement of the patient that he is unable to take quinin should constitute no bar to the use of the specific. However, cardiac depression and dyspnea are decided contraindications to the administration of the drug Happily, such cases are rare In a long experience in a highly malarial locality I encountered only two

The treatment of malaria complicating pregnancy is essentially the same as under other conditions The history of blackwater fever is no contraindication to the use of quinin While the administration of the drug is sometimes the occasion of an outbreak of hemoglobinuria, the latter is generally due to too little quinin rather than too much

The dihydrochlorid is the most valuable salt of quinin, inasmuch as it is very soluble, causing fewer nervous or gastric symptoms in the patient and being very poisonous to the parasite It is, furthermore, the most suitable preparation for intramuscular and intravenous administration The sulphate is useful in the treatment of malaria in children, being fairly palatable in suspension in aromatic syrup of yerba santa Euquinin is almost tasteless and has given satisfactory results in my hands Tannate of quinin, on account of its small proportion of alkaloid and slight solubility, has been rarely employed in this country in the therapy of malaria However, my own experience with this salt, together with the reports of the Italian physicians, leads to the following conclusions

1 The tannate of quinin is almost completely absorbed from the alimentary tract

2 It is more slowly absorbed and more slowly eliminated than the other salts of quinin, and remains in the system longer

3 A small quantity only of the salt is acted upon by the gastric juice, but is largely absorbed from the bowel after contact with the bile and pancreatic juice

4 It is not absorbed when injected into the rectum

5 It is better tolerated by the stomach, intestines, and nervous system than the sulphate

6 The clinical results with the tannate of quinin are entirely satisfactory

7 Being nearly tasteless, it is especially adapted to the treatment of malaria in children.

8 It has a good effect upon diarrhea and dysentery complicating malaria.

9 It is several times less expensive than any other tasteless preparation of quinin.

In simple cases of malaria administration of quinin by the mouth is the rule. The ideal form by this method is the solution, but the taste is so repugnant that this form has never been widely used except in hospital practice Pills and tablets are convenient to administer and not unpleasant to take, but cannot be relied upon. The coating often becomes so hard as to make solution difficult or impossible. Capsules, when fresh, are easily dissolved. The most suitable salt of quinin for injection is, unquestionably, the dihydrochlorid

The advantages of giving quinin by the needle in pernicious malaria are obviously being able to administer it to patients unable to swallow or to retain it and the certainty and promptness of absorption. The necessity of employing a dilute solution is of the first importance. Strong solutions of quinin injected into the tissues cause a wall of necrosis around the solution preventing absorption and paralyzing phagocytosis resulting, even if the solution is sterile, in nodes or ugly chemical sloughs. A solution for intramuscular injection should not be stronger than 10 per cent. The solutions should not be injected hypodermically, but intramuscularly, since in the latter location the injection is more certainly absorbed, is less apt to cause induration and abscess, and is less painful. The initial dose should ordinarily be 10 or 15 grains Afterward from 5 to 10 grains should be injected every six to eight hours as long as the symptoms demand it. The best location for injection is in the gluteal region and above the ischial tuberosities

The intravenous method is coming into more general use and deservedly so The technic usually employed is similar to that used in the intravenous injection of salvarsan However these

adept in intravenous technic may use a 10 per cent. solution of the dihydrochlorid A convenient apparatus consists of two 10-c.c. Luer syringes, connected by a stop-cock, one holding the quinin solution, the other normal salt solution As soon as the vein is entered the plunger of the salt syringe is gently withdrawn, and when blood appears in the syringe the salt solution is slowly injected to make sure that the puncture is satisfactory, in which case the stop-cock is turned and the quinin is slowly injected Some of the pharmaceutical houses are now putting out ampules containing quinin in sufficiently dilute solutions for immediate use without the addition of water or other diluent These will undoubtedly prove life-savers in the hands of many physicians who have not immediate access to facilities for preparing solutions for intravenous injection The intravenous injection should be the method of choice in the administration of quinin in cases of pernicious malaria, cases showing serious symptoms, or where there is any doubt that the drug will be retained if administered by the mouth Rectal and epidermic administration are to be regarded merely as adjuvants in the treatment of malaria

With reference to the time when the drug is given there are three chief modes of giving quinin (1) The method of Torti, a single dose before the paroxysm, (2) the method of Sydenham, a single dose in the decline of the paroxysm, and (3) the method of fractional doses The third method, that of small doses at frequent intervals, has numerous advantages over the one-dose methods

1 Quinin given in this way is better borne by the digestive and nervous systems

2 The loss of one dose by vomiting or failure of absorption is not of so much importance

3 The method is adapted to tertian, quartan, or estivo-autumnal infections, this is important, for sometimes these cannot be differentiated clinically

4 It is adapted especially to estivo-autumnal infections where sporulation is not so nearly synchronous

5 The time of administration is not dependent on parasitic

findings or definite stages, both of which may be obscure where the patient has previously taken quinin.

6 An experience in many hundreds of cases has proved its value.

I give quinin in this way almost exclusively The average dose is 1 grain an hour, given usually 2 grains every two hours, 3 grains every three hours, or 4 grains every four hours, day and night. It is especially important that the drug be given during the night, since thus only may the blood be charged during the day, when sporulation occurs Cinchonism is no guide to the quantity to be given, it is not the patient toward which the quinin is directed, but the parasite.

The specific should not be discontinued as soon as the temperature is normal, but should be kept up for at least two days longer in the quantity employed during the fever Thereafter about 15 grains on two successive days of each week should be given for at least two or three months to prevent relapse, even though the patient leave the malarial locality A few days' treatment with quinin no more cures malaria than does a few weeks' rubbing with mercury cure syphilis

As to the hygienic treatment this varies in no way from that of other acute fevers It is customary to begin the medical treatment with a purge Calomel is the drug most easily administered and retained. The dose need not exceed 5 or 6 grains and the quinin should not be delayed for the action of the purgative. During the cold stage, blankets, hot drinks, and the external applications to the head, tepid sponging, and cold rectal injections may be used. The coal tar antipyretics are not often indicated. Cold drinks may be given.

For the headache, cold applications, codein and acetanilid, or chloral and bromid of soda are useful, and if the pain demand it, morphin need not be withheld If nervousness is marked the monobromated camphor should be administered with the quinin in capsules, or the bromid of soda in solution, with each dose of the specific. For vomiting if intense and not relieved by the application of a mustard plaster to the epigastrium morphin should be employed subcutaneously

After successfully meeting the active symptoms of quinin, administered as above outlined, the prevention of a relapse is to be accomplished by giving 15 grains of quinin every sixth and seventh days for a period of not less than two or three months The administration of a valuable salt of quinin in this manner has rarely failed, in my experience, to cure the most obstinate case of chronic malaria The quinin is usually given in 3-grain doses every three hours until five are taken

Nothing is more discouraging to the physician than the treatment of cachectics in whom the poor hygienic conditions cannot be corrected, which is not rarely the case The two chief principles involved in the treatment of cachexia are first, the prevention of active outbreaks of malaria, and, second, the improvement of the general condition of the patient by appropriate hygiene Quinin is most effectively given upon two successive days in each week as described This alone, however, will rarely effect a cure except in the mildest cases

Where it is practicable a complete change of climate should be advised Without this very little can be accomplished for cases of severe degree A wholesome, nutritious, and digestible diet should be prescribed The digestion is often impaired and stomachic tonics may be indicated Exposure to inclement weather must be avoided on account of the dangers of pneumonia Occupations which subject the cachectic to violent exertion or to bodily harm should be interdicted for fear of rupture of the spleen Regular hours must be kept and constipation overcome

Of drugs other than quinin, sodium cacodylate in 3-grain doses daily have given me the best results Iron is nearly always indicated, the preparations of organic iron with manganese are best tolerated by the stomach

In the treatment of malaria there is no drug that can compare in efficiency to the salts of quinin, nevertheless, in rare instances it becomes necessary on account of idiosyncrasy to resort to the use of other remedial agents While the newer preparations of quinin—euquinin, saloquinin, aristochin, etc —are purported to be free from the toxic properties of the official salts, I have seen one case in which euquinin caused distressing dyspnea, and

another case in which this preparation caused violent urticaria. Euquinin is probably the most valuable of these preparations

Methylene blue seems to possess some specific action upon the parasites of malaria. The dose is from 1½ to 3 grams given every three hours until from 7½ to 15 grains have been given in twenty hours The patient should always be forewarned of the blue color imparted to the urine and feces

There is no doubt that salvarsan cures some cases of malaria where all other means fail Such cases are doubtless those in which "quinin fast" organisms are present. The drug should be administered by the usual technic in ordinary dosage at weekly intervals

In pernicious attacks the quinin should always be administered intravenously where this is possible, otherwise the intra muscular route must be chosen.

CLINIC OF DR ROBERT WILSON, JR.

ROPER HOSPITAL, CHARLESTON, S C

A CASE OF GONOCOCCEMIA

THE patient before us is a young white man about twenty years of age, married His occupation is that of boilermaker, and at present he is employed at the Charleston Navy Yard. He gives a history of having had the diseases common to childhood, but no other serious illness, and denies both syphilis and gonorrhea.

His present illness began about a week ago, with a rather abrupt onset. He was seized while at work with a severe pain in the lower part of the abdomen on the right side. The pain became so great that he was forced to stop work and return home. On the following day the right epididymis became involved and twenty four hours later he experienced pain in the right elbow This was followed by pain in the left knee then in the left elbow, the pain being quite intense at first The next symptom noticed was a red line extending along the inside of the left arm, from just below the elbow to above the above. The left supraclavicular glands then became affected Finally, the right knee became affected The wrists also shared in the involvement, the fingers becoming very tender somewhat painful There was no swelling of any of the joints The muscles of both extremities at this time became very painful, so that the mere weight of the bedclothes caused considerable suffering, the gastrocnemius being especially affected Two days after the onset of the illness he found that he was able to raise his hands at the wrists, the feet being similarly affected For the first three or four nights his temperature was

high, ranging from 103° to 104° F , and he was very delirious, being kept in bed with difficulty

When he entered the hospital on February 10th his temperature was 99 4° F , pulse 104, respiration 26 He was suffering very much and was given codein sulphate, ½ grain, by hypodermic During the night he was very restless and slept very little For the next two days his temperature ranged from 98° F in the morning to 100 4° F in the afternoon The pulse-rate increased progressively, but has not been above 126 at any time The respiratory rate has also increased moderately, being at present 32 His mind has been clear most of the time, but this morning he has been quite irrational Insomnia has been persistent He has been somewhat nauseated, but has not vomited Bowel movements have been involuntary for the last two days, but there is nothing characteristic about the stools The urine is scanty, it is acid in reaction, has a specific gravity of 1012, and contains a large amount of albumin, a trace of indican, a few leukocytes, and some hyaline and granular casts, there is no sugar, no acetone, and no blood Two examinations of the blood are recorded, made respectively on the morning and the afternoon of the day following admission The result of the first examination was as follows

Hemoglobin, 85 per cent , leukocytes, 34,360, small lymphocytes, 12 75 per cent , large lymphocytes, 3 per cent., large mononuclears, 0 50 per cent., polymorphonuclears, 83 75 per cent , eosinophils none, basophils none

The result of the second examination was as follows

Hemoglobin, 85 per cent , leukocytes, 54,981, small lymphocytes, 4 75 per cent , large lymphocytes, 2 25 per cent , large mononuclears none, polymorphonuclears, 93 per cent , eosinophils none, basophils none

No plasmodia were found on either examination

On examination the patient exhibits evidence of suffering much pain, and the skin and muscles of the arm, leg, and abdomen are exceedingly sensitive, the gastrocnemius is considerably swollen as well as painful The abdomen was quite distended when he was admitted, which condition has steadily

increased until the present moment, when it is very pronounced There is some resistance, especially on the right side, but this seems to be due to the superficial sensitiveness This sensitiveness renders palpation of the abdominal organs rather difficult, but there does not appear to be any enlargement of the liver or spleen There is no swelling of any of the joints, but all are very painful. Multiple neuritis is present, involving both the upper and lower extremities Wrist-drop is quite marked, but paralysis is not complete, the patient being able to raise the hand slightly with much effort. Foot-drop is less pronounced. The tendon reflexes are not abolished. The pupillary reflexes are normal There is no enlargement of the lymph-glands with the exception of those of the left supraclavicular region, which are still swollen. The epididymis is swollen and tender There is a very slight discharge of clear mucus from the urethra. The apex of the heart is in the fifth interspace, 3¼ inches to the left of the midsternal line. The valve sounds are clear and distinct, no adventitious sounds being heard at any area. The pulse is strong and full. The arteries appear to be normal. Nothing abnormal is found on physical examination of the lungs The tonsils are slightly enlarged and the mucous membrane of the fauces and pharynx somewhat inflamed. No cutaneous eruption of any kind is visible. There is no muscular rigidity

It is very evident that the patient is suffering with a severe acute infection The very high leukocyte count suggests possibly that the infecting agent might be the pneumococcus or the meningococcus, although there is an absence of any evidence of involvement of either the lungs or the meninges. The epididymitis very strongly suggests the probability that the gonococcus may be responsible for the infection although the patient denies ever having had gonorrhea, and no evidence of urethritis is present beyond a very slight mucous discharge. In order to clear up the nature of the infection a blood-culture was made by Dr Mood, who reports the presence of a Gram-negative coccus morphologically identical with the gonococcus and agglutinating with gonococcal serum, but showing no agglutination with other sera, including the polyvalent meningococcal serum of the

Rockefeller Institute He believes that there is little doubt that the organism is the gonococcus The case before us, therefore, is one of gonococcemia

Gonococcemia is a well-recognized condition The occurrence of metastases in the joints had rendered the invasion of the blood-stream evident, but the first definite proof was furnished by Hewes, who in 1894 succeeded in isolating the organism from the blood in a case of arthritis Subsequently, in 1896, Thayer and Blumer obtained a pure culture of gonococcus from the blood of a patient suffering with endocarditis, and after death were able to cultivate the organism from the vegetations on the valves Since then the gonococcus has been obtained from blood during life a number of times Faure-Beaulieu in 1906 reviewed 34 cases, and Cole has analyzed 29 cases in which blood-cultures had been made during life Loforo gives his own findings in an investigation of 67 cases Of these, "39 gave positive results" He found the gonococcus present in 19 out of 26 cases complicated with epididymitis, and in 11 out of 19 cases of chronic urethritis In 8 cases examined within the first thirty days his results were negative It is impossible to avoid the conclusion that gonorrhea becomes a systemic infection far more frequently than we have been prone to think

The portal of entrance is the affected mucous membrane. As J Aldington Gibb remarks, "there are three stages in the spread of gonococcal infection (*a*) urethral infection, (*b*) blood invasion, and (*c*) tissue invasion" Loforo believes that the organism reaches the blood-stream through the lymphatic system, but David Watson is of the opinion that while this may be true in many cases, "in others direct entry into a blood-vessel associated perhaps with venous thrombosis is the origin of the systemic infection"

It is important to note that some of the cases, as, for example, one reported by Nicoll and Wilson, showed no signs of urethritis and gave no history of gonorrhea The case before us gave a negative history, but does show some signs of a slight mucous discharge In this connection it may be noted that attention has been called to the fact that coincidentally with the onset of

acute symptoms of general infection, with chills, fever, etc., the urethritis may disappear Watson points out that "it should not require a knowledge of the presence of a concurrent gonorrhea to direct attention to the possibility of the gonococcus being the offensive agent in an obscure septicemia. The gonococcus being one of the most common disease producers, all blood examinations for unknown infecting agents should include a search for this germ"

Cases of gonococcemia may be grouped into (*a*) those whose symptoms are purely toxic, (*b*) those with metastases. The former are less frequent than the latter and may easily be overlooked In the symptoms and course they may resemble pyemia, typhoid fever, or malaria, and the diagnosis depends solely on the blood-culture. In one of Thayer's cases the patient presented "vague, general symptoms of headache, backache, and general malaise, continued fever, a thickly coated tongue, a palpable spleen, and suspicious rose spots," strongly suggestive of typhoid fever Widal tests were negative and blood-cultures showed gonococci This patient had an active gonorrhea at the time.

Of the metastases, "the most frequent is arthritis, the most important endocarditis" (Watson) Arthritis is usually polyarticular and may or may not be associated with effusion. It may happen that a history of trauma or of antecedent rheumatism is obtained, in which case these conditions probably exerted an influence in reducing local resistance, and in this way determined the point of lodgment

The association of endocarditis with gonorrhea was pointed out by Ricard as long ago as 1847, and Brandes in 1854 further emphasized this possibility Attention already has been called to the proof afforded by Thayer and Blumer that the gonococcus is undoubtedly a cause of endocarditis Most of the cases reported have been associated with arthritis, but this is not to be regarded as an essential concomitant. It does not seem to be a very frequent complication although Faure-Beaulieu found in his series only 3 cases in which there was no evidence of endocardial localization, and Cole found that in 11 cases of his series there were signs of endocarditis, and in only one of these was

the endocarditis of moderate grade In 49 cases analyzed by Kühls the aortic valve was affected 28 times, the mitral 8, and the pulmonic 6, the remaining cases showing combined lesions In several of the cases of endocarditis which have been reported pericarditis has coexisted Pericarditis alone has been reported in a number of instances

There is practically no mucous or serous surface which has not been the seat of gonorrheal infection Epipidymitis, metritis, salpingitis, peritonitis, pleuritis, meningitis, conjunctivitis, and iritis are among the more or less familiar complications

Enlargement of the spleen may occur According to Irons, it was reported to be present in 15 and absent in 4 out of 42 cases of undoubted gonococcemia The liver is very rarely involved, but icterus has been reported

The kidneys may be involved by direct extension, by metastasis, especially in the endocarditis cases, and probably in the purely toxemic cases there may be toxic albuminuria

A variety of skin lesions has been noted, including different forms of erythema Petechiæ have been observed in some cases, and in the case reported by Thayer, above quoted, a typhoid-like rose-rash was present. Others have reported similar cases Lenhartz, Thayer, and Irons have observed herpes labialis

The blood shows a polymorphonuclear leukocytosis, usually very pronounced In one of Irons' cases a leukocytosis of 40,000 was present

Multiple neuritis has been reported in some cases It may be purely toxic in origin, but not necessarily so It is usually seen in association with metastases

Delirium and stupor may likewise occur as toxic manifestations, or as the result of meningeal localization

The case before us is one of unusual interest as presenting so many of the phenomena of systemic gonorrheal infection Whether he has suffered with an attack of gonorrhea recently and disguised the fact, or whether he had a mild chronic posterior urethritis or prostatitis resulting from an old infection, cannot be ascertained It may be mentioned for what it is worth, that he has a healthy baby eight months old and that his wife has

never manifested any symptoms of gonorrhea. The beginning of the present illness may have been due to a localization in the vas deferens at the point where it curves around the deep epigastric artery, at which point perhaps there developed also a localized peritonitis The epididymis may have become involved by extension along the vas deferens from the point of the first infection Or perhaps, as suggested by Prof Phillips, the initial pain may have been reflex from a primary acute inflammation of the prostate or of the seminal vesicles Systemic infection with metastases quickly followed, arthritis, lymphangitis, and cervical adenopathy occurring as metastatic phenomena. The neuritis and delirium were probably purely toxic. The very marked sensitiveness and swelling of the muscles of the extremities and abdominal walls were probably due to diffuse myositis, a gonorrheal form of which has been pointed out by Eichhorst, Ware, and others

ATLANTA, GA.

MYOCARDIAL INFARCT FOLLOWING CORONARY SCLEROSIS

THE patient presented to you today has been in the hospital on three previous occasions—his first admission was January 15, 1916, the second admission, November 10, 1916, the third admission, July 21, 1917, and his present admission, March 18, 1918

Briefly summarized, his previous record is as follows

First admission, January 15, 1916 Mr X, white, male, married, sixty-five years of age, merchant.

Complaint, shortness of breath and cold.

Family history unimportant.

Past History —Patient states that he has always enjoyed the best of health, has had little or no sickness during his life, he remembers having had measles and pertussis in childhood, but no other of the infectious diseases He denies any venereal disease.

In 1910 he suffered with an attack of indigestion (?), which was really the first illness of any moment severe enough to make him consult a physician He states that the manifestations of indigestion which he had at this time were sour belching, regurgitation of food, and a great deal of flatulence, most noticed from one-half to one hour after eating, and an uncomfortable sensation of fulness in the epigastrium This particular ailment followed an attack of grip which did not confine him to bed He gradually recovered from the indigestion by restricting his diet, getting out of doors, drinking water, and taking exercise.

About two months after recovering from his indigestion in 1910 he was rejected for life insurance because of the presence of

albumin and casts in his urine He was told at the time that he had Bright's disease, but he did not believe it, and has received no treatment for this trouble up to the present time

Present illness began about the middle of November, 1915, when he contracted a severe cold He had some fever, with aching over his entire body, which after a few days was followed by severe paroxysms of coughing, with expectoration of very little sputum. Two or three weeks after onset he began to expectorate a considerable quantity of thin yellow tenacious material which had no odor, and was most marked in the early morning The attack lasted, with very little improvement, for six or eight weeks During the latter part of this illness he complained a great deal of shortness of breath, which would come on in nocturnal paroxysms lasting from one to two hours, the paroxysms were exceptionally aggravating and would be followed by drenching sweats and marked prostration He was not confined to bed, however, any of this time At present he suffers with marked dyspnea on exertion, attacks of nocturnal dyspnea, cough, worse at night, palpitation of the heart, and general weakness

Habits—Sleeps from 11 P M to 4 or 5 A M As a general rule he sleeps well, although of late he has been disturbed by attacks of dyspnea He eats very heavily of meat and drinks two cups of coffee daily Does not use tobacco, takes an occasional drink of whisky Appetite very good Bowels act daily without purgatives Rises from one to three times at night to urinate Works from 6 30 A M to 10 P M, with short intermissions for meals Very little or no out-of-door exercise

Physical Examination—Patient is well developed, mediumly nourished, fairly well preserved for his years Skin has a decidedly pale, pasty color, slight cyanosis of his lips and fingertips, respirations are quick, short, and shallow as he sits or reclines Temporal arteries are quite tortuous, the brachial, axillary, and temporal arteries can be seen to pulsate Respirations 36 to the minute—a bit labored and shallow Gums and mucous membranes are a trifle pale Pupils are equal, react to light and accommodation, eye movements are normal, no con-

traction of the field of vision, the retinal vessels are quite tortuous, veins distended, disks normal. Teeth are in fairly good condition. Tongue is heavily furred, protruded in the median line, breath quite foul Tonsils are not enlarged, pharynx red and congested Ears are normal There is no general glandular enlargement. No enlargement of the thyroid

Chest is well formed, barrel shaped, expansion is limited, equal on the two sides, costal angle is about 110 degrees, no bulging of the interspaces, no pulsation is observed over either the anterior or posterior portion of the chest. The percussion note over the entire chest is hyperresonant, no increase of vocal fremitus. On auscultation the breath sounds are quite distant, numerous large and medium dry râles can be heard over the entire chest.

Heart—P M I both seen and felt in the fifth is 10 cm. from the midsternal line, dulness extends to this point, no thrill is palpable. Apex beat is full and is quite forceful, no increase of cardiac dulness to the right, no increase of substernal area of dulness. At the apex both sounds are loud and snapping A^2 is decidedly louder than P^2, there is no murmur Pulse is full, bounding, 90 per minute, regular in force and rhythm, vessel wall is easily felt and quite atheromatous, systolic pressure 210, diastolic 114

Abdomen shows nothing of importance.

Genitalia normal

Extremities normal Reflexes are present, not exaggerated

Blood Examination—Red cells, 4,864,000, white cells, 6200, hemoglobin (Sahli), 95 per cent. Stained specimen showed nothing abnormal Wassermann reaction negative.

Urine—Single specimen, specific gravity 1025, decided trace of albumin, no sugar, quite a number of hyaline and granular casts.

Phthalein Test—The dye appeared in twelve minutes 25 per cent excreted the first hour, 15 per cent. the second hour

The patient remained in the hospital at rest for four weeks on a restricted diet, his condition greatly improved, all of his subjective symptoms disappearing

Second admission, November 10, 1916

Since the patient's discharge from hospital he continued to improve until October, 1916, when he states that he contracted a severe cold He states that he got quite warm while cranking his automobile, and shortly after getting back in his car he had a rather sudden pain in the left side of his chest which ran down the left arm He thinks the pain was due to riding after getting warm from the exercise which he had taken Following this he began suffering with cough, shortness of breath, expectoration of a thin mucoid material, his cough was much worse in the afternoon and evening than at any other time Occasionally during the night for the past ten days he has been awakened from a sound slumber with attacks of dyspnea, and he has suffered since the onset of this attack with considerable tightness in his chest, which he thinks keeps him from getting a long breath He tires very easily and is unable to walk any distance without great discomfort, pain, and tightness in his chest.

On physical examination at this time his respiration is 32 per minute and rather shallow Quite a number of loud, coarse, high-pitched, dry râles are heard over the entire chest and a few fine moist râles at the bases of both lungs

Heart—Apex rather diffuse, P M I in fifth is 11 cm from the midsternal line; sounds at the base and apex are clear, although there is a tendency toward muffling of the first sound The second aortic is markedly accentuated Pulse 100 to the minute, slightly irregular, due to premature contractions Blood-pressure systolic 200, diastolic 124

The twenty-four-hour output of urine was 800 c c, specific gravity 1030, albumin very heavy, microscopically contained numerous granular and hyaline casts and a few red blood-cells

On the third day of his stay in the hospital there developed over the left side of the chest posteriorly an impairment of the percussion note extending from the angle of the scapula downward over the lower portion of the chest, vocal fremitus was absent over this area, and the breath sounds were quite distant. Over the upper portion of the left lung one could hear any number of fine moist râles, the right lung was apparently normal Temperature was normal Chest was aspirated and about 200

than the previous attacks which he had had, and lasted without intermission, except for relief by the inhalation of chloroform, approximately six hours He states that had his chest been in a vise and this tightly screwed up he could not have suffered any more than he has Following the six hours of intense agony he says there was some relief in the severity of his pain, but it did not completely disappear until he commenced his journey to the hospital, when the pain suddenly disappeared, with this disappearance he was fairly comfortable for two or three hours, when his right leg suddenly became numb and he had a sensation of pins and needles sticking in his foot and the calf of his leg He states that he could walk on the leg, but that he was very much afraid of paralysis

On **physical examination** at the time the patient was admitted to the hospital he was extremely restless, tossing from side to side in his bed, markedly dyspneic, lips and finger-tips cyanosed, his expression extremely anxious, and he complained of a numbness and tingling in his right leg Pulse was 90 to the minute, regular in force and rhythm, full and bounding, systolic pressure 240, diastolic 110 The heart apex 12½ cm from the median line, forceful, and diffuse Both sounds were clear, loud, and snapping The lungs show nothing of moment.

The right leg below the knee was cold and clammy, no pulsation could be made out in the dorsalis pedis, popliteal, or in the femoral artery There was no loss of motion, no loss of strength, and sensation was intact. The patient was given his first dose of morphin at this time, which, instead of quieting him, increased his restlessness to a marked extent.

On March 19th a beginning dark reddish discoloration of the right foot was observed, this has rapidly increased from day to day until, as you see him now, the discoloration is clearly defined, involving the entire lower extremity and extending for a distance of 16 cm above the patella You will also notice that there is a marked difference between the appearance of the toes, and that portion of the leg above the knee You will notice that the toes seem to have dried up almost completely, the skin is clear and transparent and the tendons and bones are easily seen through

the skin, there is also considerable difference between the size of the feet, the right being smaller than the left The patient is still able to partially flex the right leg, but there is no motion below the knee and there is no sensation He is extremely anxious, but suffers very little except from restlessness, his respiration is quite rapid—there are numerous fine moist râles over his chest, the heart has increased in frequency, his pulse is 120 to the minute, irregular in rhythm, the area of cardiac dulness is 14½ cm from the median line, and the apex beat is quite diffuse.

Discussion —This case, followed more or less clearly for the past two years, affords you a clinical picture of the march of events and changes which occur at times in an individual with marked arteriosclerosis and atheromatous arteries, particularly where the pathologic process involves the coronary arteries We see, first, symptoms that are referable purely to the myocardium, as evidenced by his symptoms during his first stay in the hospital, his attack of influenza and probably the strain of coughing at this time was just sufficient to produce a degree of cardiac dilatation to make him consult a physician, this, in turn, was soon followed by further dilatation of his heart from unusual exertion, and with this attacks of angina pectoris An interesting feature of the anginal attacks is their frequency It is quite unusual to see an individual have as many and as severe attacks as this patient presents Then, lastly, as a result of his coronary sclerosis, he has had a thrombus form in his coronary artery which further damaged his myocardium, giving him the symptoms of which he now complains Thrombi have also formed in his heart and he has had an embolus plug his right femoral artery

Particularly do I wish to direct your attention to the symptoms of his coronary thrombosis

First, the sudden onset of violent agonizing substernal pain which persisted constantly and continuously for several hours. This particular pain differs from the pain of ordinary angina in its duration.

Second, the sudden cessation of the pain, but with its cessation

there is marked evidence that the heart has been profoundly disturbed and badly injured

Third, the evidence which makes us feel sure that such changes have occurred in the heart is that the thrombus formed within the left ventricle has been dislodged, or certainly a part of it has been dislodged, and has completely plugged the femoral artery just below Poupart's ligament.

It probably is not common to find a large superficial artery completely plugged as you see this today When such changes do occur usually smaller vessels are affected, and those of the brain most often Unfortunately, we have about reached our limit in treating this patient About all that we can do for him at the present time is to keep him as comfortable as possible and wait for the end The case is extremely difficult to handle because he belongs to that class of unfortunate individuals who cannot take morphin

NOTE —The patient lived two days after the clinic, dying very suddenly At autopsy the interventricular branch of the left coronary artery was thrombosed about 4 cm from its origin, an infarct 2 5 x 1 5 cm was found involving the tip of the left and part of the right ventricles, intramural thrombi were found in both ventricles

CLINIC OF DR JOHN P MUNROE

CHARLOTTE SANATORIUM, CHARLOTTE, NORTH CAROLINA

SEVERE HEADACHES

WE have presented a number of cases that are grouped together on the basis of their chief symptom—severe headache Some of those presented have other symptoms perhaps as prominent as the headache, and are presented on account of the diagnostic difficulties found

While one may not adhere strictly to any classification of the causes of headache, the ones usually given in text-books constitute a good working basis, which one may modify as he wishes

The general classification of the causes of headache includes [1]

1 Toxemias, exogenous and endogenous, in which impoverished or disordered blood is brought to the brain, as in (*a*) gastro-intestinal disorders, (*b*) diathetic states, gout, uremia, rheumatism, or the so-called indurative headaches, (*c*) infections, malaria, fevers, etc.

2 Toxic causes lead, alcohol, tobacco, etc.

3 Neuropathic states epilepsy, hysteria, neurasthenia.

4 Reflex causes ocular, auditory, gastric, sexual

5 Organic disease tumors, syphilis, neuritis, meningitis, and diseases of the cranial bones

Very frequently several causes act together

Headaches connected with menstruation are difficult to place in this classification They are probably both toxemic and reflex in most cases

Most of us in studying a case first make a general examination, noting any sign or symptom that may throw light on the

[1] Dana Text-book of Nervous Diseases.

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investigation We usually are gratified if we can find an organic cause, especially if it is one that can be removed The existence of a toxemia should always be carefully considered I believe the general practitioner attaches more importance to reflex causes than to any other It is interesting to note that in Cabot's graphic representation of causes the psychoneuroses stand at the head of the list He gives also a very long line to represent unknown causes I quite agree with Cabot[1] and Thomas[2] in controverting the usual text-book statement that the headaches of elderly persons are frequently caused by arteriosclerosis It is only when nephritis and high blood-pressure are present that headache in these cases may be expected It is also interesting to note that eye-strain is considered a very important cause by the general practitioner A large proportion of cases referred to me come through the oculist or are previously examined by him

CASE I —This is a woman thirty years of age, married, and has three children, the youngest child being five weeks of age at the time this patient entered the hospital, which was February 19, 1918

Her chief complaint is recurring attacks of headache associated sometimes with what her husband describes as "fainting spells" Usually the headache follows the fainting spells and frequently vomiting also occurs

The family history is negative

Past History —Patient has had two attacks of pneumonia, one twelve years ago, which was followed by severe earache. She has been practically deaf in the left ear ever since One year ago had an operation for hemorrhoids and one ovary was removed She has had three children, all normal deliveries and no miscarriages She has had marked dysmenorrhea

History of Present Illness —The headache, which is now the chief symptom, has been present more or less for the past two and a half years At first it recurred only once a month, but later became more frequent Following her last delivery, which

[1]Differential Diagnosis, vol. I
Osler's Modern Medicine, vol. vii, p 336.

was January 15, 1918, her right arm became powerless and remained so for about four days At this time also her right eye became drawn inward Since that time the so-called fainting attacks, followed by vomiting and severe headache, have recurred every few days, and she has been confined to the bed most of this time

Physical Examination.—When first seen the patient was lying on her back with eyes partially closed, mouth partially open, and the jaw fixed The arms and legs were rigid She soon came out of this condition, began vomiting, and complained of severe pain in the head, more marked in the left parietal region Her complexion was sallow and there was slight enlargement of the cervical glands Several decayed teeth had been treated by a dentist, but some pyorrhea was still present Heart, lungs, and abdomen negative Knee jerks slightly exaggerated, no clonus, no Babinski, pupils normal The eye-grounds showed the beginning of a choked disk The grasp of the left hand was slightly stronger than that of the right. Urinalysis was negative except for trace of albumin and trace of indican White blood count 8700, blood Wassermann negative. Spinal puncture showed a marked increase in spinal fluid pressure and the Wassermann of same was negative and cell count negative

We have here a group of symptoms that point rather distinctly toward a brain lesion of uncertain duration The so-called fainting spells, on investigation, proved to be a form of convulsion with a tendency to opisthotonos The history of earache, with subsequent deafness and recurring pain in the left side of her head, extending over a period of two and a half years, would suggest metastatic abscess The fact that all the symptoms became exaggerated immediately after the last child's birth would suggest that something of an acute nature occurred at that time. The choked disk and increased intraspinal pressure indicated unmistakably increased intracranial pressure, but for some time there were no distinct localizing signs. It was interesting to note that after drawing off about 30 c.c. of spinal fluid all the symptoms were very much relieved Formerly she

had been eating very little and vomited often Now she began eating regularly, seemed to enjoy her meals, and digested them perfectly At times there was slight internal strabismus of the right eye Gradually, too, there developed a noticeable paresis of the right side of the face Radiograph of the head showed an indistinct circular shadow in the left parietal region

She had been under observation about two weeks in the hospital when she had another convulsion, which was the first to occur after the spinal puncture Following this were the usual vomiting and headache and some definite localizing signs There had been some slowness and hesitancy in speech from the beginning, and this now developed into a well-marked aphasia Previously both knee-jerks had been slightly exaggerated Now the right became markedly so, and slight clonus and Babinski appeared in the right Again spinal puncture was used and the pressure was found to be somewhat above 260 As previously, all the symptoms were relieved by drawing off the spinal fluid, but the relief did not last so long as it did the first time It was found at this time, too, that choked disk was more marked and vision was distinctly diminished

Diagnosis —Considering signs and symptoms presented, I had no hesitation in making a diagnosis of a brain tumor, located in the left parietal region I advised an operation to be done, primarily to relieve the intracranial pressure and also to remove the tumor, if possible I impressed upon the husband the seriousness of the situation and told him frankly that it was quite possible the growth was malignant, in which event an operation, at best, could give only temporary relief It was natural that he should consider it for a considerable length of time before agreeing to an operation The patient grew rapidly worse, however, and spinal punctures gave so little relief that they were discontinued The convulsions became more frequent and so severe that ether was resorted to for control of them In her waking moments the patient herself began begging for an operation

On March 19th, although the patient was almost *in extremis*, Dr (Captain) Scruggs operated in the left parietal

matic pains in one knee The headache has been most severe in the afternoon and evening

Physical Examination—The patient is a stout, well-nourished man, with facial expression that indicates suffering or worry The tongue is clean, the throat normal, and there are a few decayed teeth Lungs and heart negative. Knee-jerks exaggerated, no clonus, no Babinski Pupils react sluggishly to light The right pupil is dilated The patient says his pupil has been dilated for four or five years The eye-grounds are normal Urinalysis negative except that some albumin is present White blood count 8200 Blood Wassermann positive

Diagnosis—Cerebral syphilis, with the lesions most marked at the base of the brain on the right side posteriorly There is doubtless a periostitis with a meningitis that has involved some of the fibers of the right oculomotor nerve

Treatment.—Salvarsan intravenously was begun January 25th, and given regularly once a week until five doses were given He was then given mercury and the iodids His family physician was requested to give him mercury hypodermically for a period of five weeks On account of the appearance of salivation this program was not carried out He returned to me May 29th very much discouraged and suffering again almost as much as formerly His headache was gradually relieved under the first course of salvarsan treatment, and had entirely disappeared before the fifth dose was given

In beginning the treatment again I was convinced that the intraspinous treatment should be used as well as the intravenous On May 29th I gave him 0 4 gm intravenously, and the next day gave him intraspinously salvarsanized serum to which was added 0 3 mg of salvarsan A week later I gave him another dose intravenously, but did not follow it with the intraspinous because his back was still somewhat sore Today we are giving him another dose intravenously and tomorrow will repeat the intraspinous treatment He is more encouraged about himself today than usual and says he has not felt so well in years The spinal fluid was not examined until May 30th, at which time the cell count was 540 This convinces me more than

and the selective action on one of the cranial nerves, point to luetic infection Against this is the negative Wassermann, both of the blood and the spinal fluid, and negative clinical history, so far as can be ascertained Another point in favor of luetic infection is the cell count in the spinal fluid, which is 12, considerably above normal Under the circumstances I felt justified in making a tentative diagnosis of cerebral syphilis, and expected to get further light by the therapeutic test.

Treatment —Beginning March 28th I gave her a moderate dose of salvarsan, 0 3 to 0 4 gm , once a week for eight weeks I was delighted to find that after she had received three doses the headache was very much relieved and, after the use of six doses, she needed no opiate or medicine of any kind for relieving the headache The last dose was given May 18th, and on May 20th she was able to open the left eye and now appears to be almost normal This would indicate that the diagnosis of luetic infection is correct Having completed the first course of salvarsan, we will keep her under observation for a limited period of time, and then advise that one or more courses be given, until all symptoms have disappeared and until the cell count in the spinal fluid becomes normal, regardless of the Wassermann test.

CASE IV —Male, unmarried, machinist, aged thirty-four

Family history negative.

Past History —Three years ago he suffered considerably with asthma This continued until about four months ago, when he had a growth removed from the nose, the nature of which is unknown.

Present Illness —For one year has had severe headache, especially marked in occipital region Has had frequent weak spells, during which he falls and sometimes becomes unconscious for a few minutes So far as known he has never had any convulsions For the last four months his eyesight has been gradually failing I was called to see this case in consultation with Dr Otho B Ross, the family physician, and with him I made my first examination February 24, 1918

Physical Examination —Briefly, the signs presented were as follows the pupils react to light and accommodation, partial

paralysis of the sixth and seventh nerves on the right side, with slight strabismus and occasional double vision Slight loss of sensation in both arms and hands Knee jerks absent. No clonus, no Babinski. Eye-grounds show choked disk, and examinations made later showed that it was increasing slowly Radiograph of head negative. Urinalysis showed a few hyaline and granular casts, otherwise negative Blood Wassermann negative. Spinal fluid Wassermann negative, pressure normal, and cell count normal.

Diagnosis —The symptoms and signs point very strongly to a brain lesion The interesting question is, What is its nature and where is it located? There is nothing in the clinical history nor in the laboratory test to indicate luetic infection. This may be further eliminated by the fact that the family physician had given him a specific treatment, to a limited degree, it is true, but without producing the slightest effect upon the symptoms The question of brain tumor is strongly suggested by the choked disk, severe headache, dizziness, and occasional attacks of unconsciousness It will be noticed that the sixth and seventh nerves are involved on both sides This fact and the absence of knee-jerks point very strongly to a cerebellopontine tumor The question has been raised, If there is a brain tumor, why is there no increase in the spinal fluid pressure? The answer is that the tumor in this region would probably close the aqueduct of Sylvius This would be accompanied by increase of pressure inside of the cranium and in the ventricles, but not in the spinal cavity After following this case with Dr Ross for two months and finding that the symptoms were increasing slowly, it is true, I advised an operation for decompression Perhaps a surgeon especially skilled in brain surgery would operate over the supposed seat of the tumor In this case Dr Gibbon, the surgeon called in, with Dr Ross and myself, thought a simple decompression in the temporoparietal region was the operation of choice. This would save the eye temporarily at least. If the growth should be a malignant one, no operation would be of permanent benefit. It is claimed by some that a decompression sometimes gives permanent relief, especially if the growth is of a

tubercular nature, as this kind of growth is apt to reach its maximum size, then discontinue growing, and perhaps atrophy After considerable hesitation the patient finally agreed to an operation, for the pain was becoming unbearable and the eyesight continuing to grow worse

Some time later Dr Gibbon did a simple decompression in the right parietotemporal region Patient made an uneventful recovery and for a while was very much relieved His eyesight became better, his walking more steady, and for a while there was no headache Later on symptoms of pressure gradually returned and today he is suffering as much as ever He can scarcely walk, headache is severe, vision bad, and choked disk more pronounced than ever Another decompression on the opposite side with a view to taking out a larger piece of bone than formerly is under consideration At best, however, the outlook is unfavorable

CASE V —Female, unmarried, aged twenty-two years

Family history negative

Past History —Six years ago had an acute attack of what was called "nephritis" Six months ago had whooping-cough, followed by otitis media She seems to have recovered entirely from the ear trouble Most of her life she has been subject to attacks of headache, and for the past two months it has been very severe and almost constant

Present Illness —She was referred to me on account of her severe headache, general nervousness, and frequent micturition She is an intelligent young woman, well educated, and has a tendency to introspection Appetite poor, digestion fair, bowels constipated menstruation regular and normal

Urinalysis negative except large amount of indican present. White blood count 13,200, blood Wassermann negative

Physical Examination.—General appearance, well nourished and complexion good Tongue clean, three decayed teeth have been filled, one of them having had the nerve destroyed None of the wisdom teeth have erupted Throat in good condition Heart and lungs negative Pupil reflexes normal, knee-jerks weak Cystoscopic examination of the bladder (by Dr Crow-

Present Illness—In September, 1914, he had an attack of dizziness and unconsciousness, followed by severe headache. These attacks have recurred at irregular intervals ever since, sometimes one month, sometimes several months intervening The headache sometimes precedes but more often follows the unconscious periods, and they usually come on in the morning During the last few months they have become rather more frequent, and sometimes a distinct convulsion develops

Physical Examination—He is comparatively well nourished, but there is slight atrophy of the right leg and arm His face presented a rather dull expression, voice somewhat thick, and gait slightly spastic on the right side A pustular eruption was pretty general over the body and most marked on the back. Tongue heavily coated, pyorrhea quite extensive, but no teeth badly decayed Nose and throat in good condition Lungs negative, heart presented a loud systolic murmur at the base, transmitted into the neck, a distinct thrill being felt over the same area Blood-pressure 105 S and 80 D Pupil reflexes normal Eyegrounds normal Right knee-jerk exaggerated, left knee-jerk weak Urinalysis negative Blood Wassermann negative, white blood count 7000

Diagnosis—Here is a situation where we have been handicapped by two difficulties One is the vague and indefinite history which was gotten chiefly from the patient himself The other is the fact that for a long time it was difficult to keep the patient here long enough to make a thorough examination He lives in a distant town and usually insisted on going back home the afternoon of the same day that he came to my office The fact is that, although I saw him first in August, 1917, it was not until April, 1918, that I persuaded him to stay in the hospital long enough for a real study of his case The facial expression, the unilateral spastic gait, and the exaggerated knee-jerks on the same side point to an organic lesion on the left side of the brain The history of the attacks coming on soon after a fall on the head points to a traumatism of the brain A hemorrhage with a subsequent development of a cyst around the clot is a possibility While the symptoms of irritation have increased

Present Illness—His chief symptoms I found were severe headache, pressure on top of the head, insomnia, occasional nausea, but no vomiting. Appetite good digestion fair, bowels kept open by daily doses of sal hepatica He complained of frequent accumulation of gas

Physical Examination.—His general appearance is that of a well nourished man with a troubled, anxious expression Tongue is slightly coated, teeth in good condition, several have good fillings More than a year ago radiographs were

made of the teeth, two, having abscesses, were extracted and the rest have been under the constant care of an expert dentist. Lungs are negative The heart is considerably enlarged and there is sharp accentuation of the second aortic sound Blood-pressure 240 S and 140 D Pupil reflexes normal, knee-jerks slightly exaggerated Urinalysis sp gr 1018, albumin present in large quantity, sugar negative, bile negative, indican excessive, many hyaline and granular casts and epithelial cells White blood count 10,000 Wassermann negative

Diagnosis —With the above history, signs, and symptoms one would have little difficulty in making a diagnosis of chronic nephritis The patient's circumstances were such that he could not remain in the hospital for treatment at the time of the first examination He promised to go home, however, arrange his affairs, and return as soon as possible

When he entered the hospital five weeks ago practically the same conditions existed as already described The urinary output by the red test was about 60 per cent of normal With rest in bed, milk diet, sweat baths, and elimination by the bowels his headache and insomnia have been largely relieved The blood-pressure has been reduced very little

Prognosis and Discussion —The outlook is serious, and my experience is that one in this condition seldom lives one year, more often it is a matter of a few months The surprising thing is that, although he has been under the care of several most excellent physicians, the serious aspects of this case were either not recognized or not taken firmly in hand until too late for any treatment to be of much avail A persistent headache with high blood-pressure always calls for thorough investigation with reference to the kidneys In this case, as in many that I have seen, undue attention seems to have been directed to his eyes and errors of refraction

Proper and intelligent care was given to his teeth Even granting that a thorough investigation of the kidneys would not have disclosed any organic change eighteen months ago, still the conditions, as outlined, were so suggestive that he should have had repeated examinations and careful oversight all the time

It was a serious mistake to allow him for nearly a year to continue in the full work of a pastorate where the duties were known to be large and exacting

CASE VIII.—Man, aged twenty nine, unmarried Mill operative. This patient was first seen seventeen days ago, and his chief symptom was severe pain in the left parietal region

Family history negative

Past history negative

Present Illness—He dates his illness from April 1, 1918, when he had a very severe case of influenza Following this an otitis media developed in the left ear The membrane ruptured and the pain disappeared, a tolerably profuse discharge resulting Ten days later he had severe pain in the left parietal region He was in Indianapolis at that time and fell into the hands of a competent surgeon He made a diagnosis of "abscess of the brain" and advised an operation This the patient refused Later his friends brought him back to his North Carolina home and sent him to me for examination

I found him still suffering with severe pain over the left side of the head, marked hebetude, slowness of speech, and evidence of beginning aphasia

Physical Examination.—Pale, somewhat emaciated, tongue clean, pupillary reflexes normal, teeth carious, no enlargement of cervical glands The arm jerks were normal, knee-jerks weak, co-ordination good, no Romberg, no clonus, no Babinski. Heart and lungs negative Hearing good and equal in both ears, both cords normal, no choked disk (Dr Peeler) Urinalysis faint trace of albumin, trace of indican White blood count 11,200 Blood Wassermann negative. Patient was advised to come to hospital as soon as possible for further examination and probable operation He entered the hospital one week ago and the symptoms of brain pressure had increased considerably since the first examination His headache was more severe and his mental condition almost stuporous. He complained of not seeing well and often seeing double. Right knee-jerk exaggerated, clonus and Babinski also present in the right Left knee-jerk slightly exaggerated, slight clonus, no Babinski

During this second examination a spinal puncture was made and there was slight increase of pressure, Wassermann negative, cell count 10 polymorphonuclear, 2 mononuclear (Dr Barret)

Radiograph (Dr Lafferty) —"The transverse view shows very much clouding around the mastoid cells on the left side and the auditory canal is larger than normal, as if some of the bone had been removed The mastoid cells are practically obliterated and the dense area extends upward much higher than normal Just above this region is a translucent area that may be a cyst or abscess, although it does not seem to have clearly defined walls The sella turcica is also abnormal and it seems as if it were filled with some dense material that projects forward"

Diagnosis —The history of this case clearly indicates that the beginning of this trouble is to be found in the influenza and the middle-ear disease, occurring about the first of April Although there is no special tenderness about the mastoid cells, the *x*-ray examination indicates disease there which may call for operative treatment later There is evidently a lesion in the left parietotemporal region involving the speech center It would appear also that there is some lesion at the base of the brain involving the sella turcica

The diagnosis made in Indianapolis was "abscess of the brain above the ear" The history of the case would exclude acute abscess, but it may very reasonably be a chronic abscess The *x*-ray examination suggests a cyst This is quite possible as a result of a hemorrhagic encephalitis, resulting from a nonsuppurative infection from influenza or otitis media. In either event it appears that there is a diseased condition both at the cortex of the left side and at the sella turcica The cortical lesion is now demanding immediate operation, so at my request Dr Scruggs operates today We are expecting to find either a chronic abscess or a cyst developed upon a hemorrhagic encephalitis

Operation.—After making the usual incision, Dr Scruggs removed an oval-shaped piece of bone about 1¼ inches in its longest diameter The membrane immediately bulged out into the opening A crucial incision was made in the dura and a cyst

was disclosed about the size of a large plum. The cyst was opened and its contents discharged, but the area was too vascular to justify any attempt at removing the cyst wall

Prognosis —This operation should give at least temporary and possibly permanent relief, so far as the cortical lesion is concerned As there appears to be a lesion at the sella turcica, it is quite possible that serious trouble may result from it later on.

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